

adjusted at 5 L/min & systemic resistance at 20 mmHg/L/min
Mean ABP (diastolic + $\frac{1}{3}$ pulse p) will be 105 mmHg refer to vasc.

Systemic venous filling p = MSFP - RAP = 5 mmHg

Cardiac output (curve) is affected by 5 factors:

Preload, Afterload, Inotropic state, HR and heart size

1 Venous filling pressure (Preload) i.e. MSFP - RAP

During diastole, ventricle is very compliant so $\oplus\oplus$ preload

$\rightarrow \oplus\oplus \text{ EDV} \rightarrow \oplus\oplus \text{ SV (Starling law)} \rightarrow \oplus\oplus \text{ COP}$

Factors $\oplus\oplus \text{ EDS}$

- 1 $\uparrow$ Atrial contraction
- 2 $\uparrow$ Total blood volume
- 3 Venous tone by symp. stim
- 4 Pumping by sk ms cont.
- 5 $-ve$ intrathoracic p

Factors $-- \text{EDV}$

- 1 Standing
- 2 $\oplus\oplus$ intrapericardial p.
- 3 $--$ vent. compliance
e.g. myocardial infarction

2 After load = Aortic & large arteries p (mean ABP)

End-systolic p. (ESP) nearly equals aortic p so it represents Afterload

$\oplus\oplus \text{ Afterload} \rightarrow \oplus\oplus \text{ ESV} \rightarrow -- \text{ SV (Starling law)} \rightarrow \ominus \text{ COP}$

So, cardiac output curve is shifted downward and to the Rt.

3 Inotropic state of heart i.e. contractility

Contractility not dependent on pre or afterload i.e. intrinsic capacity

$+ve$ inotropic stimuli shift systolic pressure-volume curve upwards

and shifts COP curves upwards & Lt & vice versa

Mechanism $+ve$ inotropic $\rightarrow -- \text{ ESV} \rightarrow \oplus\oplus \text{ SV} \rightarrow \oplus\oplus \text{ COP}$

Factors affecting inotropic state of whole heart (intrinsic)

A Intrinsic factors

1 Force-Frequency relation $\oplus\oplus$ frequency of contraction (26)

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Electrocardiogram

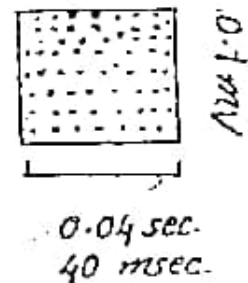
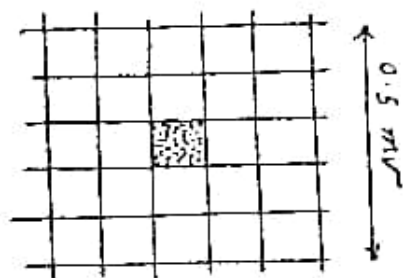
ECG

Definition

record of rapid voltage change of heart.
Biphasic action potential.
from the skin Body fluids are good conductor.

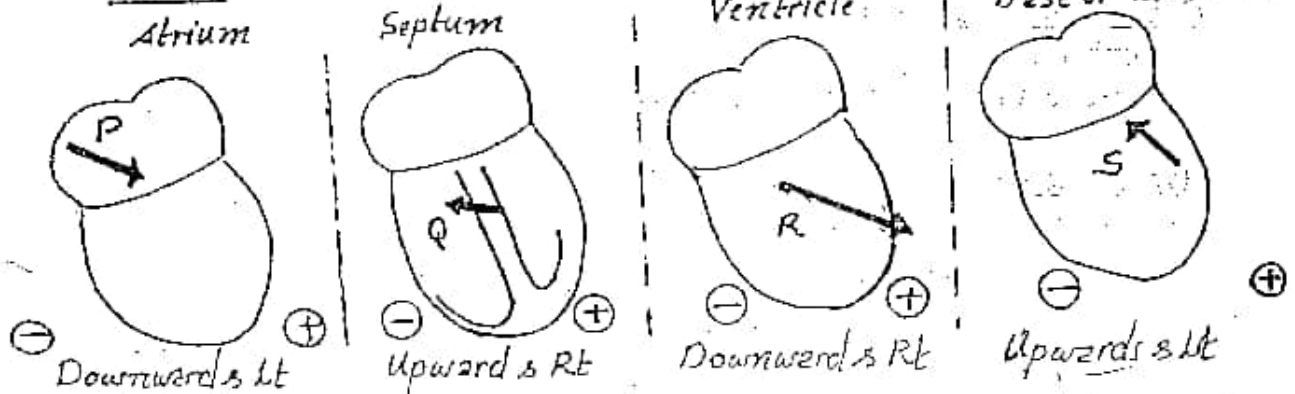
Recording

- Apparatus : 1. Monitoring machine ICU
2. Recording machine fed to heart stylus pen recorder
- Calibrated strip of paper



25 small square / second
1500 " / minute

Vector :



Voltage $\propto$ mass of tissue

Duration $\propto \frac{1}{\text{velocity of conduction}}$

Polarity

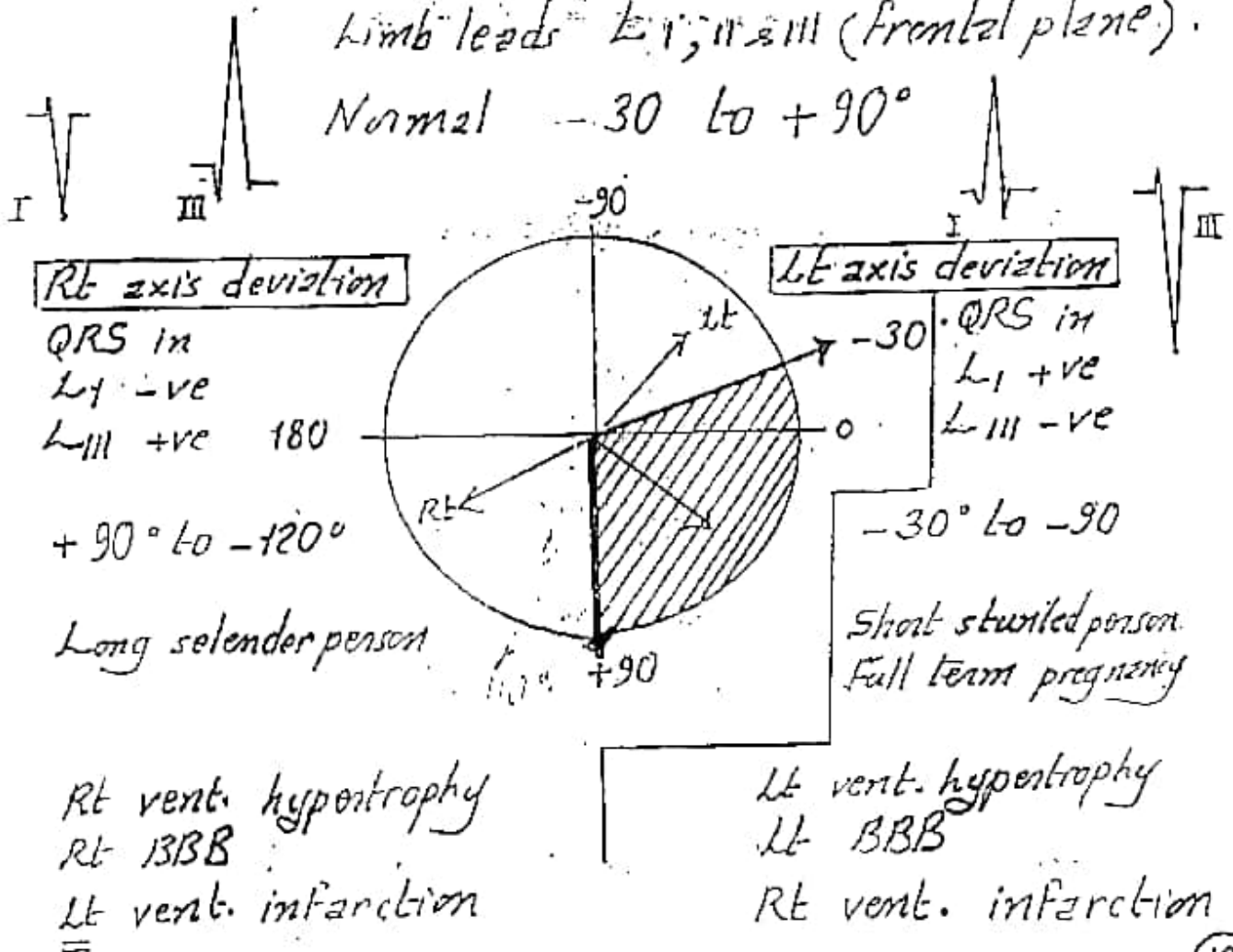
Depol is directed towards +ve electrode
 —————→ positive wave (upward) & vice versa
 Repol is directed towards +ve electrode
 —————→ negative wave (downward) & vice versa (10)

1 Heart rate HR $\frac{1500}{\text{number of } \square \text{ between 2 following R}}$
 normal 60 - 90 average 75/min.

2 Rhythm almost regular
 i.e R waves at regular intervals

3 Voltage i.e voltage of QRS in limb leads
 normal $\geq 1.6 \text{ mV}$ i.e 16 mm.

4 Axis of the heart i.e vector of QRS in unipolar limb leads I, II, III (frontal plane).
 Normal -30 to $+90^\circ$



Electrocardiogram ECG

Definition Record of rapid voltage changes of heart

2 skin electrodes i.e. Biphasic AP. Body fluids are good conductor

Recording of ECG:

1 Apparatus a Monitoring machine viz. CRO used in IUC

b Recording machine fed to heat stylus open recorder

on a moving calibrated strip of paper. 

Small square 40 msec i.e. 25 squares/sec = 1500 squares/min

Small square 0.1 mV.

Vector = an arrow represents sum of electrical activity

Vector represents

Direction of vector

Atrial activity

Downwards & to Lt

Septal activity

Upwards & to Rt

Vent. activity

Downwards & to Lt

Base of Lt vent

Upwards & to Lt

Length of vector

$\propto$ mass of tissue

Duration

$\propto$ $\frac{1}{\text{velocity of conduction}}$

Polarity

If Dep. is directed towards +ve electrode $\rightarrow$ +ve wave

If Repol. is directed " +ve " $\rightarrow$ -ve wave since

If vector is parallel to lead $\rightarrow$ maximal effect

perpendicular to lead $\rightarrow$ not recorded

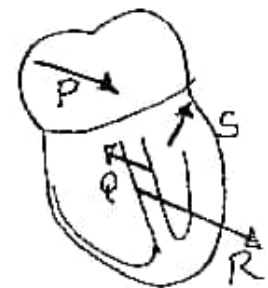
Lead position of the 2 electrodes or actual recording

According to TYPE of electrodes, leads may be:

1 Bipolar leads between 2 exploring electrodes (-ve & +ve)

e.g. Standard limb leads

2 Unipolar leads between one exploring electrode (+ve electrode) and one indifferent -ve electrode conducted by connecting simultaneously to the 3 limbs (Rt arm, Lt arm & Lt foot) to ① resistance 500 ohm.



e.g. 1 Augmented limb leads One +ve electrode attached to a limb, the other electrode is attached to the other two limbs. Potential is 50% more without affecting configuration of the record.

2 Unipolar chest leads precordial leads V_1 to V_6

3 Esophageal lead rarely used to record post. surface of atria

N.B. Rt leg is considered earthing electrode.

V (unipolar) a (augmented) L (Lt arm) R (Rt arm) F (Lt foot)

[A] Bipolar leads (standard limb leads)

	Exploring -ve electrode	Exploring +ve electrode
1 Lead I	R	L
2 Lead II	R	F
3 Lead III	L	F

[B] Augmented unipolar limb leads

	Exploring +ve electrode	Other electrode
1 aVR	R	LF
2 aVL	L	RF
3 aVF	F	LR

[C] Unipolar limb leads

	Exploring +ve electrode	Indifferent electrode
V_1	Rt 4th space parasternal	RLF
V_2	Lt 4th space parasternal	RLF
V_3	between V_2 & V_4	RLF
V_4	5th space midclavicular line	RLF
V_5	5th space ant. axillary line	RLF
V_6	5th space mid axillary line	RLF

N.B. Einthoven triangle heart at its centre

Approximated by putting electrodes at R, L & F.

Zero potential if the 3 electrodes are connected to indiff. electrode (2)

Normal ECG

Intervals = Waves + Segments

Waves	P	qRs	T	U
Represents	A. depol.	V. depol.	V repol.	Papillary m. repol
Duration (width)	0.08 - 0.15 i.e 80 - 100 msec	0.08 second	0.16 second	0.05 sec.
Voltage (height)	- Limb leads 0.25 mV - Chest leads	1 mV 3 - 4 mV	> 0.5 mV > 1 mV	Usually not recorded
Shape	1st half for Rt atrium 2nd half for Lt atrium	V ₁ , V ₂ small R, large S V ₃ , V ₄ Moderate R, S V ₅ , V ₆ large R, small S	Slightly rounded & asymmetrical	
Direction	Inverted in aVR	R always +ve Q -ve before R S -ve after R	Inverted in aVR	

NB - A. depol.: masked by QRS complex & T very low voltage.

- Depol. of SAN, AVN & Purkinje f. not recorded (small mass of tissue).

- Q wave Depth $< \frac{1}{4}$ (1-2mm) Width < 0.04 sec.

Pathological Q wave i.e large Q wave indicates myocardial infarction

Q normally appears in Lead I, aVL, V₅ & V₆

Disappearance of growing R from V₁ to V₆ = serious vent. condition

Variation in voltage of QRS helps in diagnosis of vent. enlargement.

Increased duration of QRS helps in " of bundle branch block.

- T wave (repol) in same direction of R wave (depol) because last part to be depol. (epicardium) is the 1st part to be repol.

During systole, compression stress -- bl supply & delays repol. in subendoc

Segments

- P-R segment 0.08 to 0.1 second
Measurement From end of P to beginning of QRS

(3)

Vector is algebraic sum of A. potential at a point.
 Each P wave, QRS complex & T wave has a vector.
Normal calculated axis of heart is -30° to $+90^\circ$

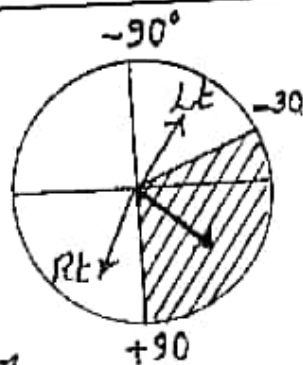
Rt axis deviation

QRS -ve in L I
 +ve in L III

Axis from $+110^\circ$ to -90°

Physiol. Long slender person

Pathol. Rt vent. hypertrophy (RVH)
 Rt bundle branch block (RBBB)
 Lt. vent. infarction



Lt axis deviation

QRS +ve in Lead I
 -ve in Lead III

Axis from -30° to -90°

Short stunted person

Full term pregnancy

Lt. vent. hypertrophy (LVH)

Lt. bundle branch block (LBBB)

Rt. vent. infarction

N.B Relationship of vent. monophasic A.pot. to ECG:

QRS coincides with rapid depol.

T wave " " rapid repol

ST segment " " plateau

Relationship of cardiac contraction to ECG:

P wave occurs before the beginning of atrial cont. by 0.02 s

QRS complex " before " " ventricular cont. by 0.02 s

Ventricle remains contracted until after end of T wave

ECG abnormalities

A) Disorders of rhythm

1. Simple sinus tachycardia

HR 100-150/min

Fever $0.5^\circ\text{C} \rightarrow \begin{cases} ++8/\text{min} & \text{adult} \\ ++10-15/\text{min} & \text{child} \end{cases}$

Symp stim.

A-V nodal rhythm (if SAN is damaged)

P inverted or absent & short P-R interval

Simple sinus bradycardia

HR below 60/min

Athletes at rest (high vagal tone)

Vagal stim. in carotid sinus syndrome

regular 200-350/min	ectopic focus discharges	irregular 300-500 per min
P: QRS 2:1 or 3:1 i.e. incomplete heart block		P waves are absent & replaced by F waves
QRS at regular intervals		QRS at irregular intervals

4 Vent. extrasystole Vent. premature beat (VPB)

Cause Ectopic vent focus discharges occasionally due to excessive smoking, coffee drinking or alcohol & lack of sleep or abdominal distension

ECG P absent
QRS giant, bizarre and prolonged.
T inverted
Compensatory pause follows VPB

B) Disorders of conduction

1 Atrio-ventricular block i.e. lesion in A-V bundle.
1st degree All atrial impulses are conducted to ventricle but with difficulty prolonged P-R interval

2nd degree Some impulses are conducted others do not
Atrial beats (P) : Vent. beats (QRS & T) 2:1 or 3:1

3rd degree (Complete heart block) No atrial impulses are conducted
Ventricles beat independent of atria at slow rate
i.e. idioventricular rhythm 35-45/min

There is complete dissociation between P waves and QRS & T

2 Bundle branch block (BBB) Lt or Rt bundle lesion

Excitation passes through muscle to activate vent. on blocked side

Vent rate normal QRS complexes are prolonged & deformed

C) Low voltage ECG i.e. normal summation of QRS complexes
in L_1 , L_{II} or L_{III} $< 1.5 \text{ mV}$ ($< 15 \text{ mm}$)

Causes Obesity (body mass index > 30)

or hypothyroidism (myxedema)

High voltage ECG sum of QRS in L_1, L_{II} or L_{III} > 3.5 mV (> 35 mm)

D) Lt vent. hypertrophy (LVH)

Lt axis deviation

S in V_1 or V_2 + R in V_5 or V_6

35 mm in adults

45 mm in children

Rt vent. hypertrophy (RVH)

Rt axis deviation

Reversal of precordial pattern i.e.

- tall R in V_1 & V_2

- Deep S in V_5 & V_6

E) Myocardial infarction

Cause -- bl. supply to part of myocardium irreversible changes and death of muscle cells

1 Acute myocardial infarction

Elevation of S-T segment of infarcted area in the 1st hour.

2 After some days or week S-T elevation subsides

Pathological Q ++ in size and width of Q wave

F) Electrolyte imbalance

1 Hypokalemia Flattening or inversion of T wave & prominent U wave

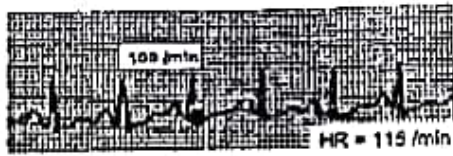
Hyperkalemia Tall, thin T waves

2 Hypocalcemia prolonged Q-T interval.

Hypercalcemia shortening Q-T interval.

Abnormal ECG

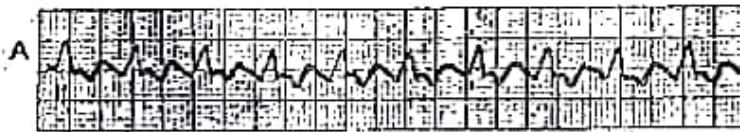
A) Disorders of rhythm



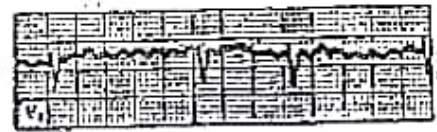
sinus tachycardia $> 100/\text{min}$



sinus bradycardia $< 60/\text{min}$



Atrial Flutter A 2:1 B 4:1



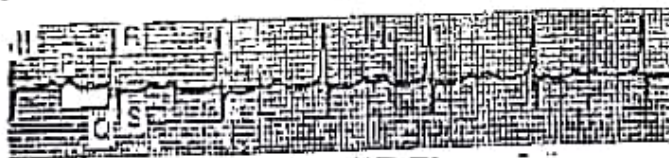
A. fibrillation



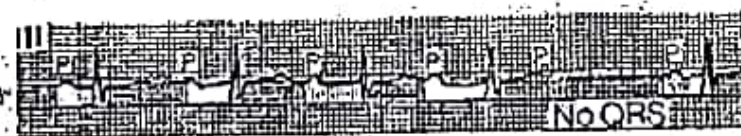
Ventricular extrasystole (PVBs)

B) Disorders of conduction

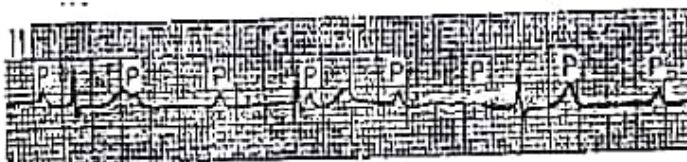
A.V block i.e Heart block



1st degree

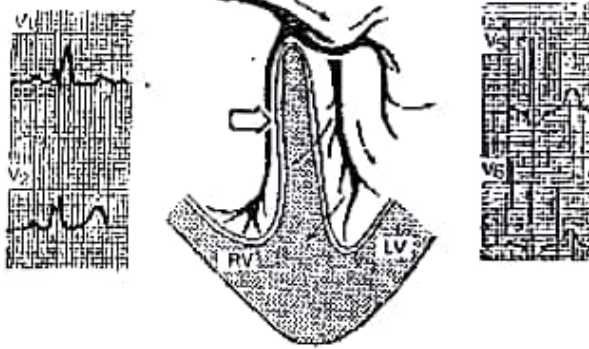


2nd degree
Wenckebach



3rd degree
Complete heart block

RT BBB (V_1 & V_2)



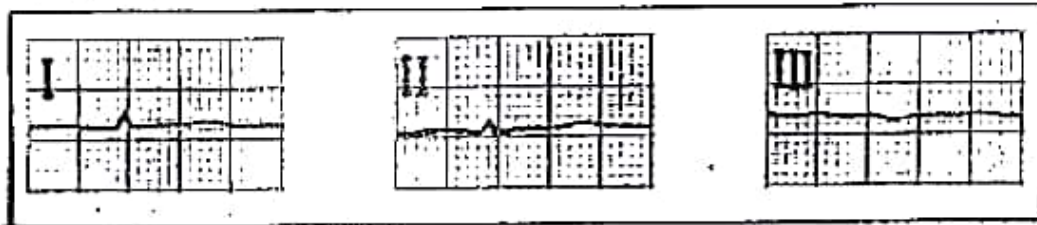
Delayed RV activation
Double hump QRS

LT BBB (V_5 & V_6)



Delayed LV activation
Double hump QRS

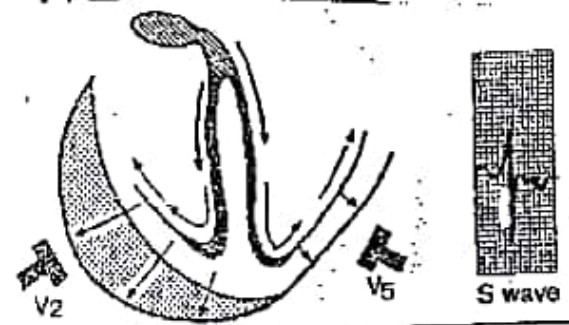
C) Low voltage ECG e.g. hypothyroidism (myxedema)



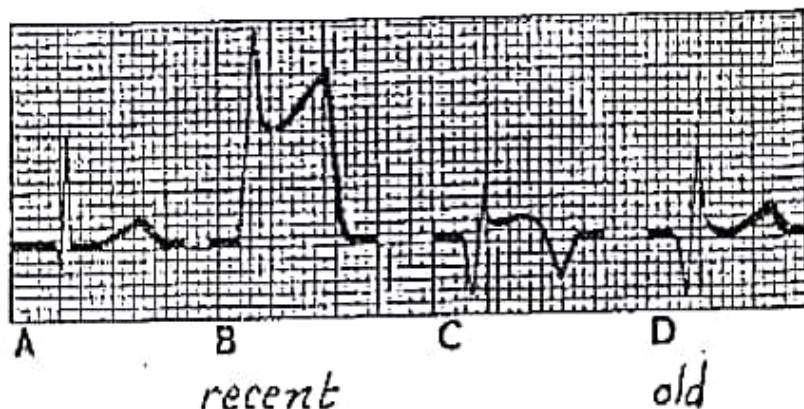
D) Lt. ventricular hypertrophy



Rt ventricular hypertrophy

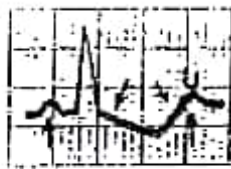


E) Myocardial infarction

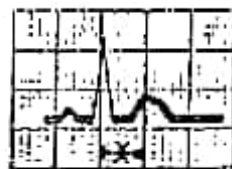




Hyperkalemia



Hypokalemia

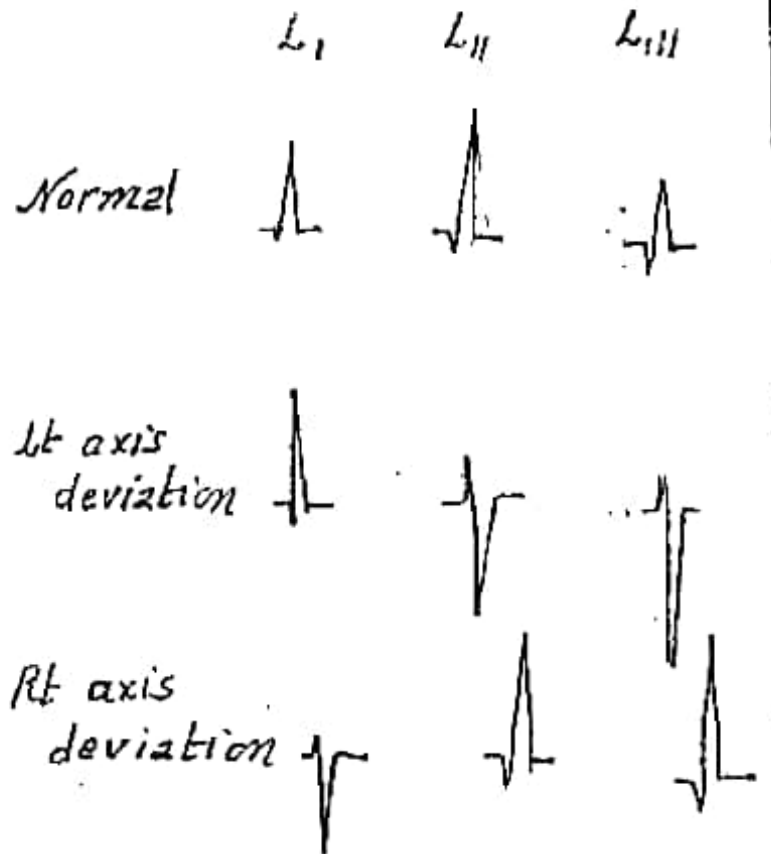
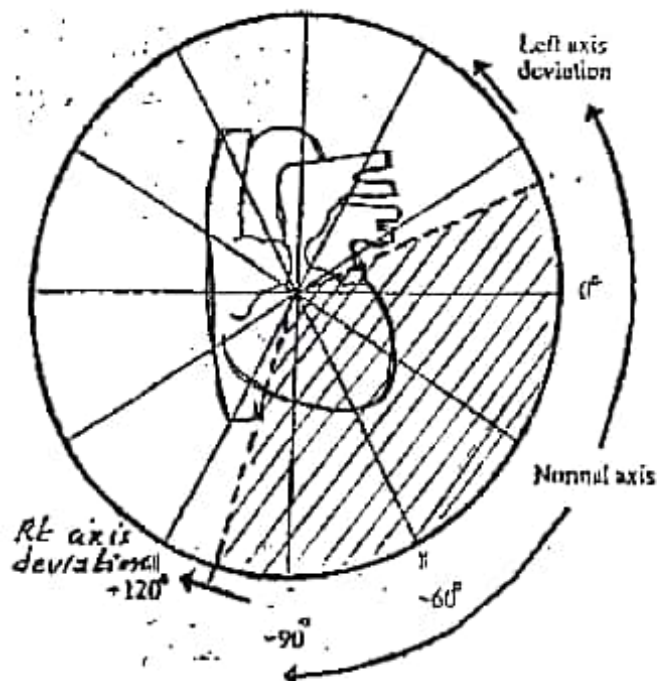


Hypercalcemia



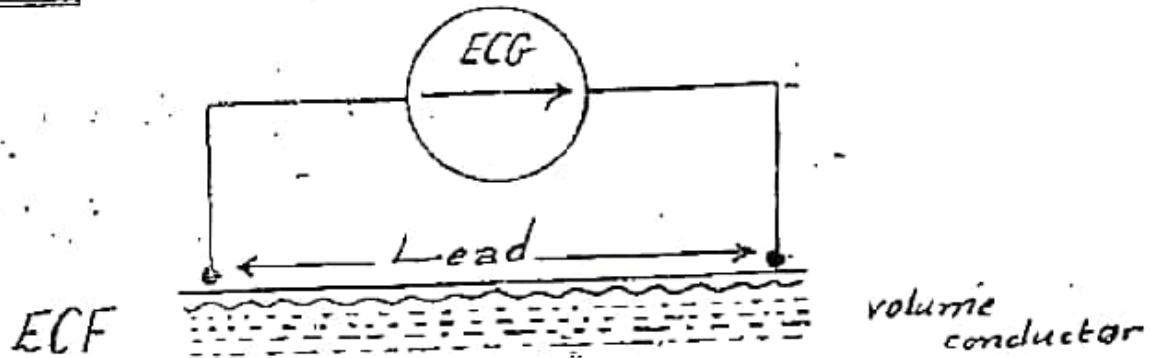
Hypocalcemia

E) axis deviation



Vector is parallel to lead $\rightarrow$ maximal effect
 perpendicular to lead $\rightarrow$ not recorded

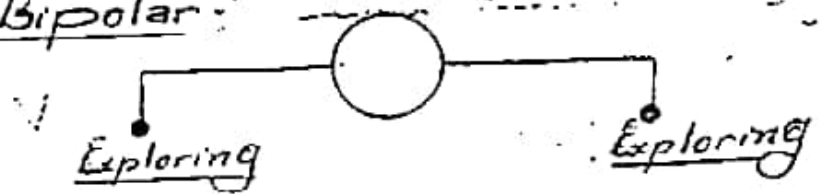
Lead : Position of the two electrodes



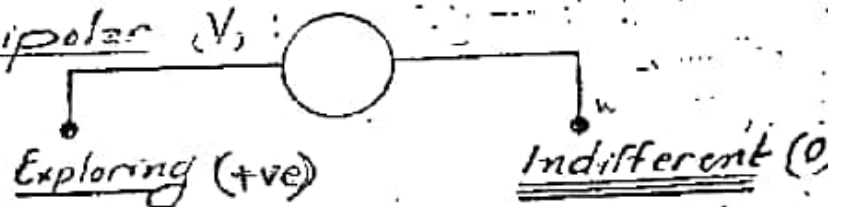
Types of leads :-

(A) According to TYPE of electrodes :

① Bipolar



② Unipolar V₁



(B) According to SITE of electrodes :

① Limb

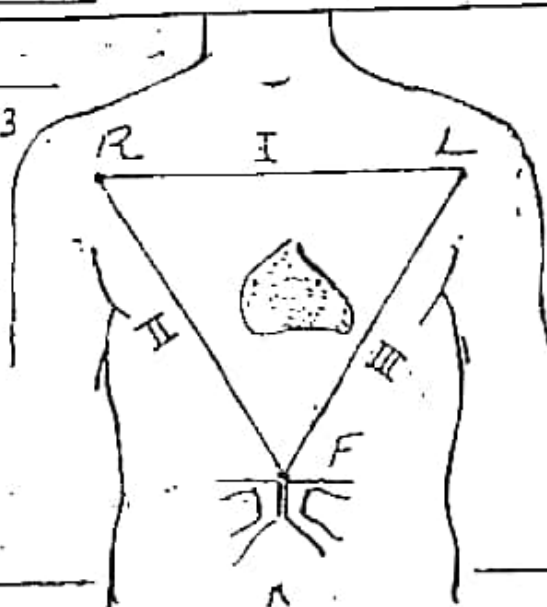
Recording in FRONTAL plane

② Chest

Recording in HORIZONTAL plane

Unipolar leads

- Augmented limb 3
 aVR
 aVL
 aVF
- Chest δ V₁ to V₆
- Esophageal rarely used



Bipolar leads

- Limb 3
 I
 II
 III

Cardiac cycle

Period From beginning of one H. beat to beginning of next beat
A-V delay (0.15) allow Atrial cont. emptying before Vent. cont.
C. cycle consists of a period of relax (diastole) & cont. (systole).

During diastole: Heart rest, coronary bl. flow to subendocardium of Lt. vent occurs and most of ventricular filling occurs.

	HR 75/min.	HR 200/min
Duration of C. cycle	0.8	0.3
„ Vent. systole	0.3	0.15
„ Vent. diastole	0.5	0.15

If heart rate $\oplus\oplus$, diastole is shortened more than systole.

HR up to 160 as in exercise leads to increased cardiac output

Above 160/min, filling is decreased and cardiac output decreases.

C. cycle 8 phases & 8 changes look table page

Notes Some regurgitation of bl. into veins occurs during atrial systole.

Vent. systole starts by cont. of intervent. septum pulling on fibres around the A-V valves closing them & resulting in 1st heart sound.

Late in systole, aortic p exceeds vent. p. for a short period. Momentum (kinetic force) keeps blood moving forwards.

Main function of atria is bl reservoir & not to pump blood; because 70% of vent. filling occurs passively. So, atria are not essential for life.

Pressure - volume graph of Lt ventricle

4 phases

Phase I Period of Filling

During diastole, The ventricle fills passively

- Filling pressure For Lt. vent is pulmonary venous pressure
For Rt. vent is RAP i.e. central „ „
= 5 mmHg (between 3 & 7 mmHg) (19)

• Vent. volume increases to 130 ml i.e EDV called preload
Phase II Period of isovolumetric contraction.

- Vent. pressure rises from 0 to 80 mmHg for Lt vent.
- Vent. volume is kept constant (130 ml); as vent. contracts isovolumetrically & moves from diastolic to systolic compliance curve.

Phase III Period of ejection

- Vent. pressure exceeds aortic p. & aortic valve opens
 - " " rises to a maximum of 120 mmHg then it $\ominus$
 - Vent volume decreases from 130 to 50 ml (ESV)
- Contraction changes suddenly from isometric to isotonic

Phase IV Period of isovolumic relaxation

At end of ejection, aortic valve closes & vent relaxes isometrically. R. moves vertically from systolic to diastolic compliance curve.

- Vent. pressure falls to zero i.e below atrial p. & mitral valve opens
- Vent. volume is kept constant (50 ml) i.e ESV

N.B From curve, Stroke volume = $EDV - ESV = 130 - 50 = 80 \text{ ml}$

Arterial pulse

Blood forced into aorta sets up a pressure wave which expands the arterial wall as it travels. Expansion is palpable as pulse.
Velocity of p. wave 7 meters/sec. while bl velocity in aorta 0.5 m/s.
In elderly, arteries are more rigid so pulse wave moves faster.

The pulse is felt in radial artery at wrist 0.1 s after ejection peak.
Strength of pulse $\propto$ Pulse pressure i.e Systolic P - Diastolic P.
Pulse is weak (thready) in shock & strong in exercise.

Atrial pressure changes

Atrial p. changes are transmitted to great veins e.g. jugular vein (JV) producing 3 pressure waves

c wave is due to rise of atrial p during isometric vent. con.
v wave is due to rise of atrial p before AV valve opens during diastole

Heart sounds HS

Four HS

Two sounds are normally heard by stethoscope during cardiac cycle:

	First heart sound	Second heart sound
<u>Cause</u>	Vibrations set up by the A-V valves	sudden closure of Semilunar valves
<u>Time</u>	Diastole end (start of systole)	Systole end (start of diastole)
<u>Best heard at</u>	Apex of heart (5th Lt space midclavicular (mitral comp.)) Lower end of sternum (tricuspid component)	Second Rt costosternal junction (aortic component) Second Lt intercostal space parasternal (pulm. component)
<u>Characters</u>	<u>Longer</u> 0.15 second isometric contraction & max ejection. <u>Low pitch</u> 25-45 Hz <u>LUB</u>	<u>Short</u> 0.12 second isometric relaxation <u>High pitch</u> 50 Hz <u>DUB</u>

A.B In male (mitral area) 10 cm (3.5 inch) from middle line below Lt nipple.
Physiologic splitting of 2nd HS: During inspiration, time between aortic & pulm. valve closure is long enough for 2nd HS to be reduplicated.
 Time between 1st & 2nd HS = Systole & between 2nd & 1st HS = Diastole.
Two HS are normally non-audible by stethoscope.

Third heart sound	Fourth heart sound
May be heard in <u>young</u> during diastole	<u>Rarely</u> heard in normal adult
Coincides with <u>rapid filling</u> (rush of bl.)	Due to vent. filling during <u>A. systole</u>

Murmur Abnormal noise sounds heard over the heart

Mechanism Change of bl. flow from laminar flow (silent) to turbulent flow (creates sounds) This occurs when blood exceeds a critical velocity e.g when a heart valve is narrowed.

Major cause is valvular diseases

1 Atrial systole

- 2 Isovolumetric contraction
- 3 Maximal (rapid) ejection
- 4 Reduced ejection

5 V. diastolic

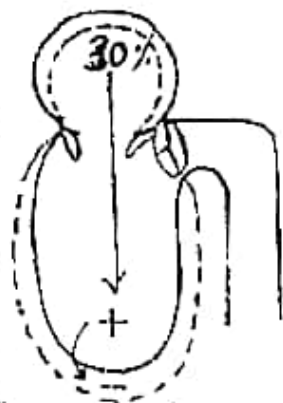
- 6 Isovolumetric relaxation
- 7 Maximal filling
- 8 Reduced filling

Ventricular Systole 0.3 second

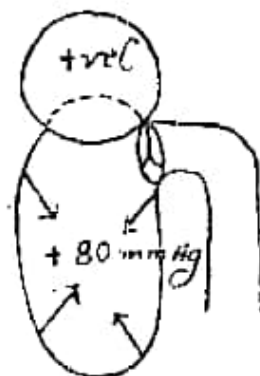
2, 3 & 4

Ventricular Diastole 0.5 second

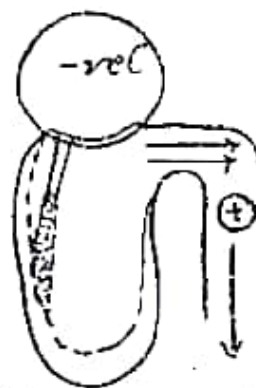
<u>Early</u>	<u>Mid</u>	<u>Late</u>
5, 6 & 7	8	1



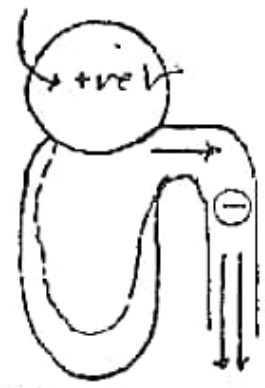
A. systole



Isovolumetric cont.



Maximal ejection



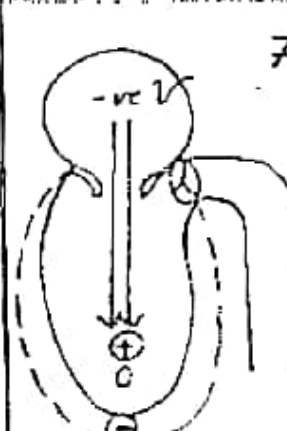
Reduced ejection



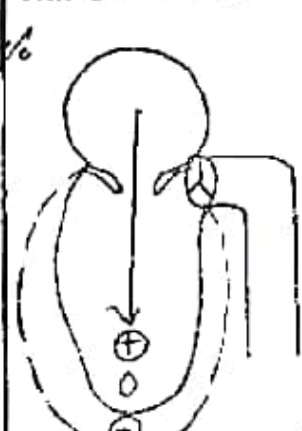
Protodiastolic



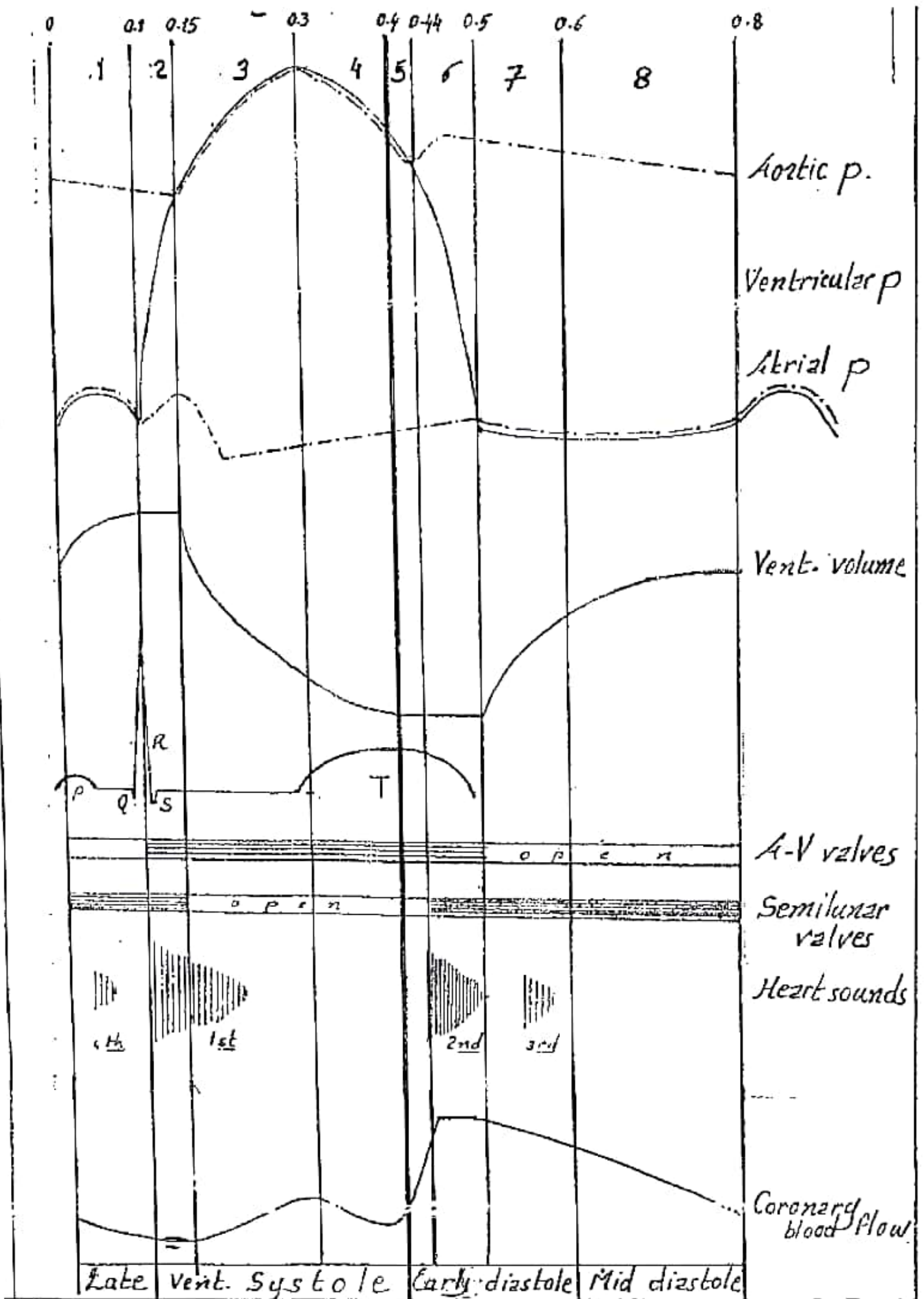
Isovolumetric relax.



Maximal filling



Reduced filling



Cardiac output

Definitions

- Stroke volume Vol. of bl. pumped by one ventricle per BEAT
 $SV = \text{end-diastolic vol.} - \text{end-systolic vol.}$ $SV = EDV - ESV$
- COP (minute vol.) Vol. of bl. pumped by one ventricle per MINUTE
 $COP = SV \times HR = 70 \text{ ml} \times 70 \text{ beats/min} = 5 \text{ Litre/min}$
- Cardiac index COP per square meter surface area $= 3.2 \text{ L/min/m}^2$

Measurement

1 Direct Fick method

Fick principle $F(\text{flow}) = \frac{\text{Amount of substance taken by organ}}{\text{Arterio-Venous (A-V) difference}}$

$$COP (\text{of Rt vent.} = \text{Lt vent.}) = \frac{O_2 \text{ consumption in mL/min}}{A_{O_2} - V_{O_2}}$$

$$= \frac{250 \text{ mL/min}}{190 \text{ mL/L} - 140 \text{ mL/L}} = \frac{250 \text{ mL/min}}{50 \text{ mL/min}} = 5 \text{ L/min}$$

Arterial O_2 content is measured in a sample taken from any artery
Venous bl. sample is taken from pulm. artery by cardiac catheter

2 Indicator dilution technique

Indicator : Dye (cardiogreen) or Radioactive isotope is injected IV
Not harmful, has no haemodynamic effect & not excreted rapidly

$$CO = \frac{\text{Amount of indicator}}{\text{Average conc. of indicator}} \times \frac{60}{\text{Time of first passage}}$$

Average conc. of indicator & time of 1st passage are known from the concentration-time curve by plotting the log of indicator conc. against time in a serial arterial samples
Log of indicator conc. is used to eliminate big variations in concent.

The conc. of indicator rises, falls then rises again due to recirculation.

3 Thermodilution technique - same principle of indicator dilution. (23)

Indicator : Cold saline injected in Rt atrium by double lumen catheter

in the other longer side of the catheter.

Advantages 1 Saline is completely harmless.

2 No second rise i.e. recirculation is not a problem.

4 Doppler combined with echocardiography, non-invasive technique.

Various conditions that affect COP

<u>Increased</u>	<u>Decreased</u>	<u>No change</u>
<u>Excitement & anxiety</u> 50-100%	<u>Sitting or standing</u>	<u>Sleep</u>
<u>Exercise</u> up to 70%	<u>From lying</u>	<u>Moderate change in</u>
<u>Eating</u> 30%	20-30%	<u>atmospheric temp.</u>
<u>Exposure to high atmosph temp.</u>	<u>Rapid arrhythmia</u>	
<u>Epinephrine</u>	<u>Heart diseases</u>	
<u>End of pregnancy</u>		

Factors affecting performance of whole heart (same as isolated ms)
i.e. preload, afterload, inotropic state and heart rate.

Control of cardiac output

2

1 Venous return: which is primarily affected by peripheral circulation

In the steady state: $VR = COP$

2 Cardiac performance.

Venous return curves & COP curves explain the interplay between 2 factors:

Venous return

Guided by Ohm's law $Flow (F) = \frac{\text{Pressure gradient } (\Delta P)}{\text{Resistance } (R)}$

$$VR = \frac{MSFP - RAP}{RVR} \quad \text{i.e. 3 factors affecting VR}$$

1 MSFP Mean Systemic Filling Pressure

2 RAP Right Atrial Pressure (Central Venous Pressure (CVP))

3 RVR Resistance to Venous Return

(24)

1] Mean Systemic Filling Pressure (MSFP)

Def. P in systemic circulation when blood flow is stopped. or simply P in veins as capacity of veins is 18 times that of arteries. It can be measured in heart-lung preparation if the pump oxygenator which replaces the heart is stopped.

Normally 6 - 8 mmHg. It represents distending p. of all vessels
MSFP $\propto \frac{\text{volume of blood}}{\text{vol (capacity) of vascular system}}$

2] Right Atrial Pressure (RAP) i.e central venous pressure (CVP)

Venous return (Venous function) curves:

VR is plotted against RAP (RAP normally 0-2 mmHg)

$$\boxed{VR \propto \frac{1}{RAP}}$$
 refer to curve

• Down slope ++ RAP $\rightarrow$ -- VR VR = 0 when RAP = MSFP = 7 mmHg

• Plateau occurs as RAP becomes less than -1 mmHg

-- RAP $\leftarrow$ ++ ΔP $\rightarrow$ ++ VR
collapse of intrathoracic veins $\rightarrow$ -- VR so VR $\oplus$

• Transitional zone between down slope & plateau

Increased blood vol or venoconstriction shifts VR curve upwards (Lt)
i.e increased VR & vice versa

3] Resistance to Venous Return (RVR)

++ RVR shifts VR curve downwards (Lt) i.e -- VR & vice versa

$$RVR = \frac{MSFP - RAP}{VR} = \frac{7 - 0}{5} = 1.4 \text{ mmHg/L/min.}$$

Most of RVR occurs in veins; accordingly sudden drop of COP & fainting occurs if VR -- by mild compression of inferior vena cava.

Cardiac output curve:

COP is plotted against RAP

$$\boxed{COP \propto RAP}$$
 refer to curve

- [2] Ischemia (—bl flow) produces -ve inotropic effect due to ↓ O_2 acidosis.
- [3] Chronic myocardial failure → -ve inotropic state.

[B] Extrinsic Factors

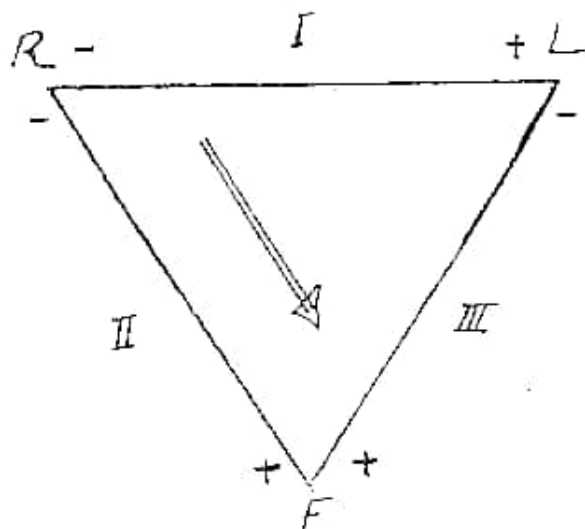
- [1] Activation of symp. adrenergic nerve → +ve inotropic.
Mechanism Norepinephrine via β_1 receptors → cAMP which activates pln kinase A → phosphorylates voltage gated Ca^{++} channels → ++ opening time of Ca^{++} channels → accelerates systole
 → ++ active uptake of Ca^{++} by SR → " diastole
- [2] Activation of parasymp (vagal) nerve → -ve inotropic mainly on atrial muscle via acetylcholine.
- [3] Xanthine e.g. caffeine -- breakdown of cAMP → +ve inotropic
- [4] Glucagon ++ formation of cAMP → +ve inotropic
- [5] Digitalis (cardiac glycosides) inhibits Na^+-K^+ ATPase
 → ++ intracell. Na^+ → inhibits Na^+/Ca exchanger
 → ++ intracell Ca^{++} → +ve inotropic
- [6] Increased extracell Ca^{++} conc. +ve inotropic (not in sk ms)
- [7] Ca^{++} antagonists (DHP) & antiarrhythmic drugs -ve inotropic

N.B. Heterometric autoregulation Homeometric autoregulation
Preload Regulation of COP by changing Inotropic state

[4] Heart rate affects COP in triphasic manner

- [a] Changes in HR within physiological range 50-160/min have little effect on COP; as it is corrected by opposite changes in SV. This is because VR governs COP in normal heart
- [b] At higher rates, COP ⊖ because of shortening of filling time
- [c] At lower rates, COP ⊖ because SV is maximal and can't compensate for reduced HR below 50/min.

● Bipolar limb leads 3
i.e. Standard limb leads



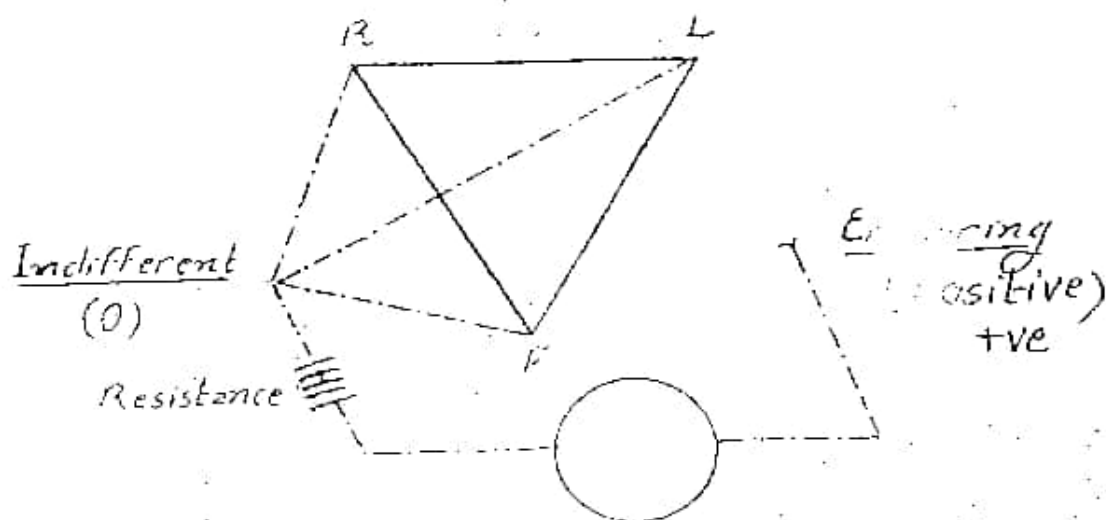
$R = Rt \text{ arm}$

$L = Lt \text{ arm}$

$F = Lt \text{ foot}$

note
Rt foot earthing

Unipolar lead 9 V



$$\text{Indifferent} = (R - L) + (L - F) + (F - R) = 0$$

Exploring $\begin{cases} \text{Limb} & \text{Unipolar limb leads} \\ \text{Chest} & \text{Unipolar chest leads} \\ \text{Esophagus} & \text{Unipolar esophageal lead} \\ & \text{rarely used} \end{cases}$

Cir (12)

++ HR by symp. stim. as in exercise $\rightarrow$ -- duration of A. potential & systole $\rightarrow$ more time for filling $\rightarrow$ ++ COP 204mm at HR 200/min because of ++ MSFP +ve inotropic effect.

4 Heart size (Hypertrophy) 2 types

a Eccentric (volume overload) b Concentric (pressure overload)

Trained athletes	e.g	Hypertension
Increased	EDV (H. size)	Constant
Mild thickening	Myocardium	Marked thickening
May over 35 L/min	COP	Less
Normal	Inotropic state	Heart failure if not lt
		++ lt vent. systolic p

Equilibrium between VR and COP

Equilibrium point occurs at intersection of VR curve & COP curve
i.e. $COP = VR$. At rest, 2 curves intersect at Rt atrial p 0-2mm
Mechanism Starling law allows COP to equal VR.

Assessment of contractility by Ejection Fraction (EF)

Definition Fraction of EDV ejected each beat.

Measurement non invasively by cardiac ultrasound (ECH)

Normally About 0.6 in a healthy heart

Factors affecting EF Contractility also filling p & aortic p

Significance Most important index of contractility

if below 0.5 suggests cardiac disease.

if below 0.3 is associated with high mortality.

Selected integrated responses

• Supine to erect posture Pooling of bl. in LL veins

-- MSFP $\rightarrow$ -- VR $\rightarrow$ -- COP $\rightarrow$ -- ABP

Compensated by reflex ++ HR, ++ contractility & ++ ABP
Both VR & COP curves shift upwards

Phase	Ventricular Systole				Early diastole		Mid diastole
	1 Late diastole	2 Isometric contraction	3 Maximum (rapid) ejection	4 Reduced ejection	5 Proto diastolic (isovolumic) relax.	6 Maximum filling	8 Reduced filling
Change	1. A-systole	2 Isometric contraction	3 Maximum (rapid) ejection	4 Reduced ejection	5 Proto diastolic (isovolumic) relax.	6 Maximum filling	8 Reduced filling
1 Ventricular Pressure	0.1 (+) due to A-systolic then due to V. diastolic	0.05 (+) to 80 mmHg Lt V. 10 mmHg Rt V. ends by opening of semilunar valves	0.15 (+) to max. 120 mmHg Lt V. 25 mmHg Rt V.	0.1 (-) Reduced ejection	0.04 (-) Proto diastolic (isovolumic) relax.	0.06 (-) to 0 mmHg Lt V. 0 mmHg Rt V.	0.1 Around 0 mmHg Vent. relax = vent. filling (-) = (+)
2 Ventricular Volume	Increased > 100% (30% in 1st)	Constant	Decreased rapidly	Increased slowly	Constant	Increased rapidly	Increased slowly
3 Aortic Pressure	Ca, La, Co, Aortic	Blood leaves aorta to peripheral vessels	Ascending Aorta starts to eject in mid 1st leaving 120 mmHg	Descending Aorta starts to eject in mid 1st leaving 120 mmHg	Constant	Increased rapidly	Increased slowly
4 Atrial Pressure	+ve & -ve (Atrial closure of ventricles of ventricles)	+ve (A-V closure)	-ve (Decline)	-ve (Decline)	-ve (Decline)	-ve (Decline)	-ve (Decline)
5 A-V valves	Open	Open	Open	Open	Open	Open	Open
6 Semilunar	Open	Open	Open	Open	Open	Open	Open
7 Heart Sounds	4th HS vent. closing by A-systole	1st HS sudden closure of A-V valves long low pitch	2nd HS	3rd HS	4th HS	5th HS	6th HS
8 ECG	P starts 0.025 before A cont	QRS starts 0.025 before V. cont	T wave begins	T wave peaks	T wave ends	T wave ends	T wave ends
9 Coronary bl. flow	MINIMUM	MINIMUM	Slight ++ with aortic P	Slight -- with aortic P	MAXIMUM	MAXIMUM	MAXIMUM

- 1 Heart energy 99% aerobic 1% anaerobic $\rightarrow$ ++ to 10% in exercise
- 2 So, cardiac muscle has more
blood supply, myoglobin & mitochondria (30% of Cms vol)
- 3 Cardiac ms has NO O_2 debt
- 4 Cardiac ms utilizes more substrates
FAT 60%, CHO 35% & ketone and aa 5%

Myocardial O_2 consumption MVO_2

TEC Total chemical energy $\propto MVO_2$

Basal (non beating) MVO_2 2 ml & beating MVO_2 9 ml / 100g/min

N.B Since cardiac venous O_2 tension is low, increase in O_2 consumption requires increase in coronary blood flow.

Myocardial O_2 consumption is determined by:

- Systolic p. (wall tension)
- Shortening extent (stroke vol)
- Inotropic state contractility
- Heart rate.

Cardiac work Work output of heart

$$\text{Work} = \text{Load } (P) \times \text{Distance } (\Delta L)$$

$$\Delta L = EDV - ESV = SV$$

Systolic load is an index of afterload (P_s)

Work of Lt ventricle

Work of Rt ventricle

$$= SV \times \text{mean aortic } p$$

$$= SV \times \text{mean pulmonary } p$$

Energy converted to work each BEAT is STROKE work output.

Energy converted to work each MINUTE is MINUTE work output.

Forms of cardiac work output

- 1 Volume - pressure work or external work 98% of C. work

$$= SV \times \text{Pressure}$$

For Lt it is 6 times that of Rt vent.

Used to move blood from low p. veins to high p. arteries

i.e Work performed by ventricle to raise p of blood each beat (30)

afterload (pressure work) + preload (volume work)

O_2 consumption/beat is determined by the area which represents:

a External work ejection loop plus

b Internal work triangle between systolic & diastolic curve.

It represents additional work output if vent. empty all its bl. content

2 Kinetic energy of blood 2% of total work.

Work used by heart to accelerate velocity of bl. in aorta & pulm.

Cardiac efficiency

Efficiency = $\frac{\text{Cardiac work/min i.e. External or effective work}}{MVO_2}$

MVO_2 represents total energy (determined by SV, HR, systolic P & cont.)

Normally, Maximal 20-25%. In heart failure 5-10%.

Cardiac reserve CR

Definition Maximum % COP can increase above normal

Normal young adult : up to 300-400%.

Athletic trained person : 600%.

Elderly : 200%.

Heart failure : 0%.

Involves 1 Factors ++ preload (EDV) 2 Factors -- afterload (aortic P)
3 +ve inotropic stimulants 4 +ve chronotropic stimulants
5 Hypertrophy

Diseases that -- CR 1 Ischemic heart disease.

2 Myocardium damage (infarction) 3 Valvular heart disease.

Diagnosis of low CR (Exercise test)

Method: Treadmill or walking up & down steps

In low CR COP fails to maintain body activity.

Test is cutoff if 1 Shortness of breath (dyspnea)

(31)

- 2 Muscle fatigue resulting from muscle ischemia.
- 3 Maximal increase in HR ($220 - \text{Age in years}$)

Heart Failure

Definition: Failure of heart to pump enough bl to satisfy body needs

Manifestations:

- Forward : -- COP
- Backwards : Lt ventricle pulmonary congestion
Rt ventricle systemic congestion

Causes :

- ① Systolic dysfunction e.g myocardial infarction $\rightarrow$ inefficient cont.
 - ② Diastolic dysfunction e.g hypertension $\rightarrow$ -- diastolic compliance
- In both cases, EF is low down to 20%.

Response to -- COP in HR :

- 1 ++ symp. discharge.
- 2 ++ circulating catecholamines.
- 3 ++ afterload $\rightarrow$ more -- in COP.
- 4 VC of renal vessels $\rightarrow$ Na^+ & H_2O retention.
- 5 ++ renin $\rightarrow$ ++ angiotensin II $\rightarrow$ more VC
- 6 ++ aldosterone $\rightarrow$ more Na^+ & H_2O retention.
- 7 ++ ANP due to ++ RAP $\rightarrow$ Na & H_2O excretion (escape phenomenon)
- 8 Visceral congestion

Edema is due to end organ unresponsiveness to ANP.

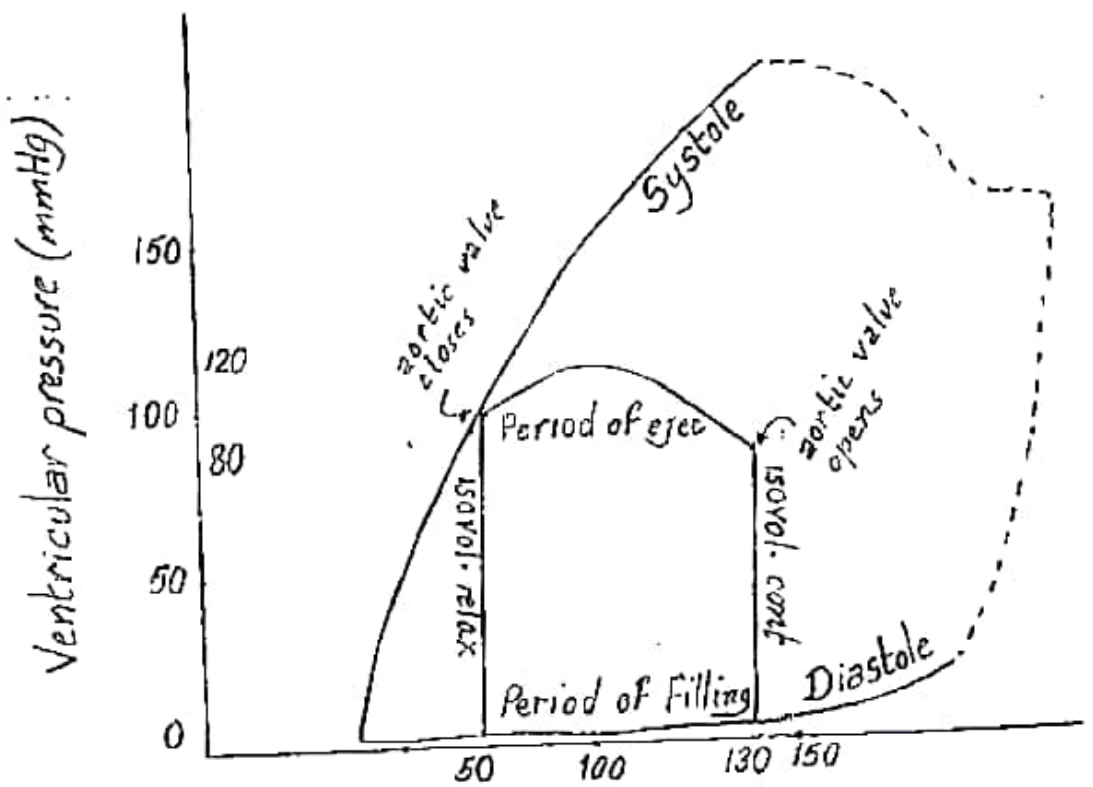
ttt 1 +ve inotropic e.g digitalis

2 Diuretics $\rightarrow$ -- preload.

3 Arteriolar vasodilatation $\rightarrow$ -- afterload

4 ACE inhibitor $\rightarrow$ -- Angiotensin II $\rightarrow$ -- afterload
 $\rightarrow$ -- Aldosterone $\rightarrow$ -- preload

Pressure-volume graph (loop) of Lt ventricle



Cardiac output

Stroke volume
SV

Minute volume
Cardiac output
COP

Cardiac index
CI

by one ventricle

Volume of bl. pumped

$$\text{best } SV = \frac{EDV - ESV}{130 - 60}$$

$$\text{Minute } COP = \frac{HR \times SV}{72 \quad 70}$$

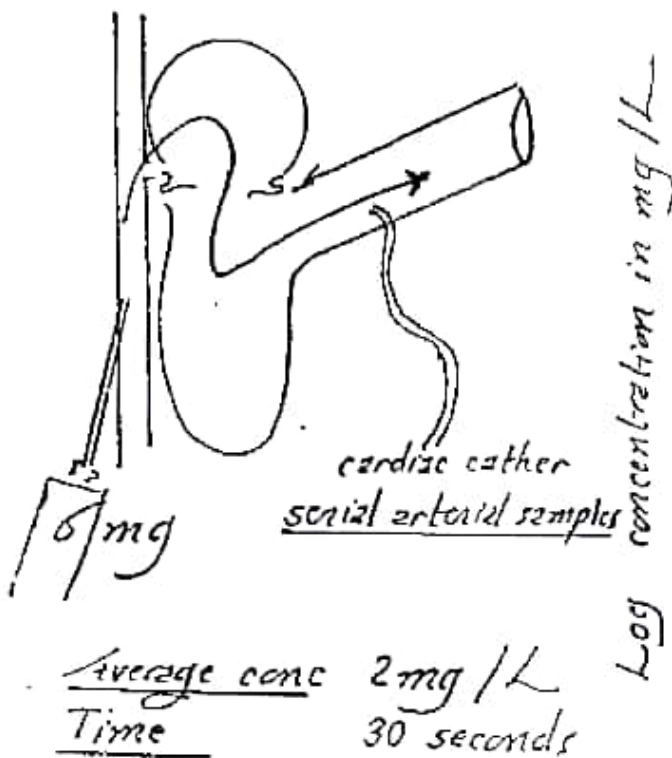
$$\text{Minute/sq m } \frac{5/1.7}{3.2 \text{ L/m}^2/\text{m}}$$

① Indicator dilution technique

Indicator Dye (cardiogreen) or Radioactive isotope

NOT $\left\{ \begin{array}{l} \text{toxic} \\ \text{excreted very rapidly} \\ \text{affecting haemodynamic i.e. no VC or no VD} \end{array} \right.$

$$COP = \frac{\text{Amount of indicator}}{\text{Average conc of indicator}} \times \frac{60}{\text{time of 1st passage}}$$



② Thermodilution technique

Indicator Cold saline

Advantages

- Saline is harmless (not toxic)
- No second rise

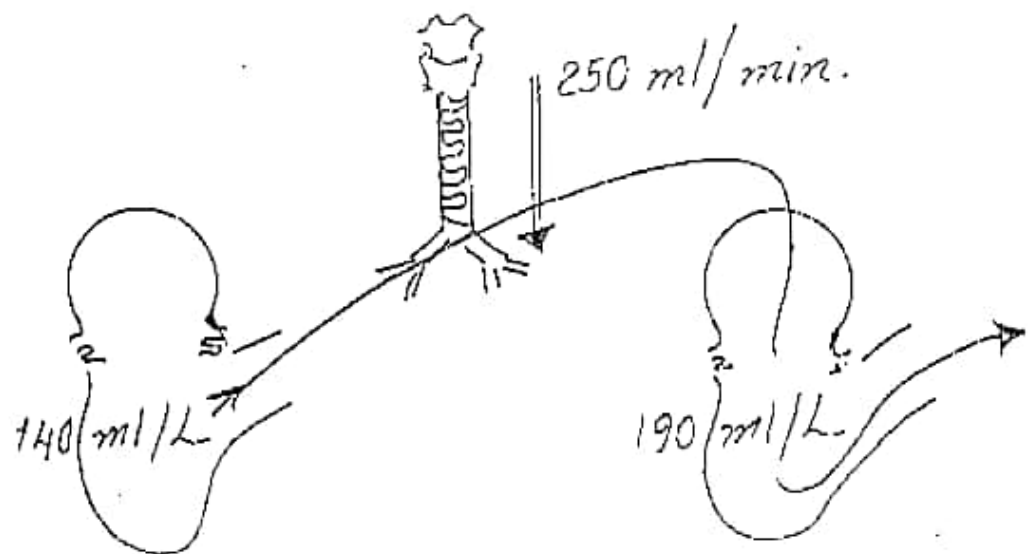
③ Other method

- Doppler combined with echocardiography
- Direct Fick method.

Direct Fick method

$$F = \frac{\text{Amount of substance taken / min}}{\text{Arterio-venous difference}}$$

$$\begin{aligned} \text{COP} &= \frac{\text{O}_2 \text{ consumption (ml/min)}}{\text{Arterio-venous O}_2 \text{ difference}} \\ &= \frac{250 \text{ ml/min}}{190 \text{ ml/L} - 140 \text{ ml/L}} \end{aligned}$$



Conditions that affect COP

±	Sleep	moderate temp changes
+	Excitement	upto 100%
	Exercise	70%
	Eating	30%
	Epinephrine	
	Hot atmosphere	
	Pregnancy	
-	Lying	to standing
	Rapid arrhythmia	
	Heart diseases	

- 1 VR primarily determinant
- 2 Cardiac performance

Under normal conditions, $COP = VR$ within limit of Cardiac performance

Factors affecting VR

Ohm's law
$$F = \frac{\Delta P}{R}$$

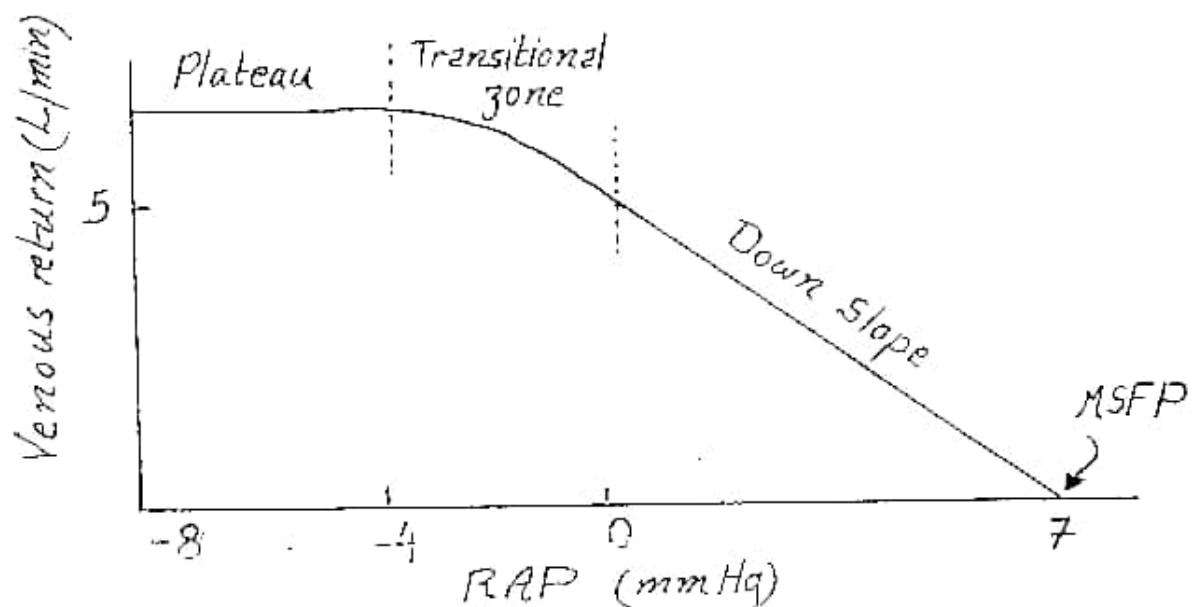


$$VR = \frac{MSFP - RAP}{RVR}$$

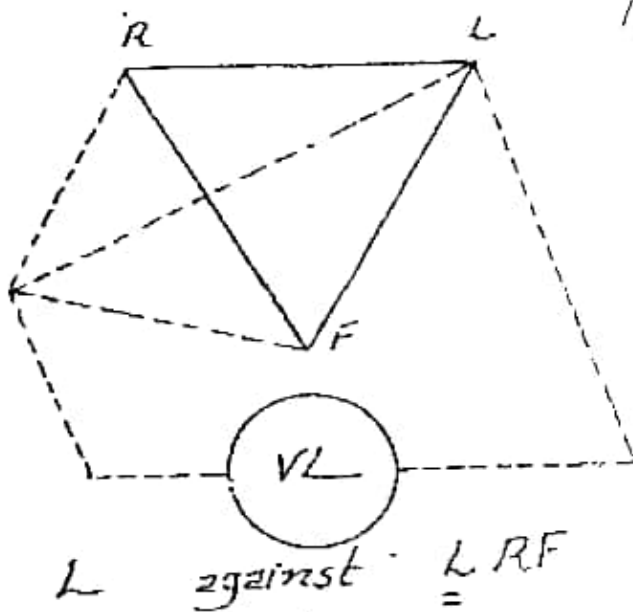
1 MSFP 6-8 mmHg $\propto \frac{\text{Volume of blood}}{\text{Capacity of vascular system}}$

2 RAP (CVP) 0-2 mmHg $VR \propto \frac{1}{RAP}$

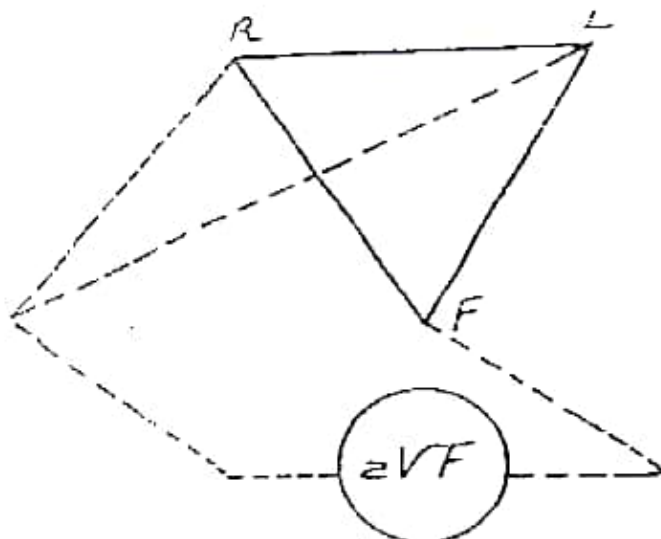
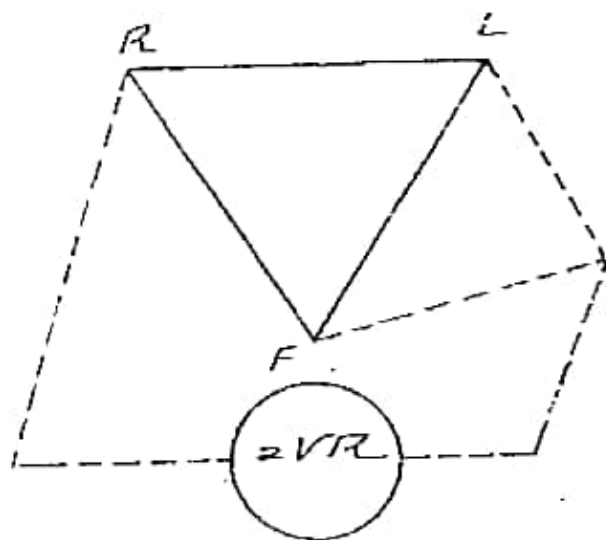
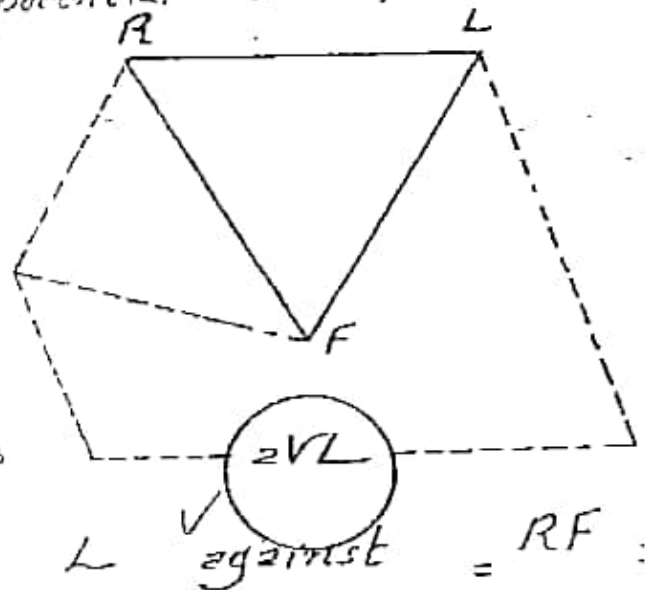
Venous return (Venous Function) curves :



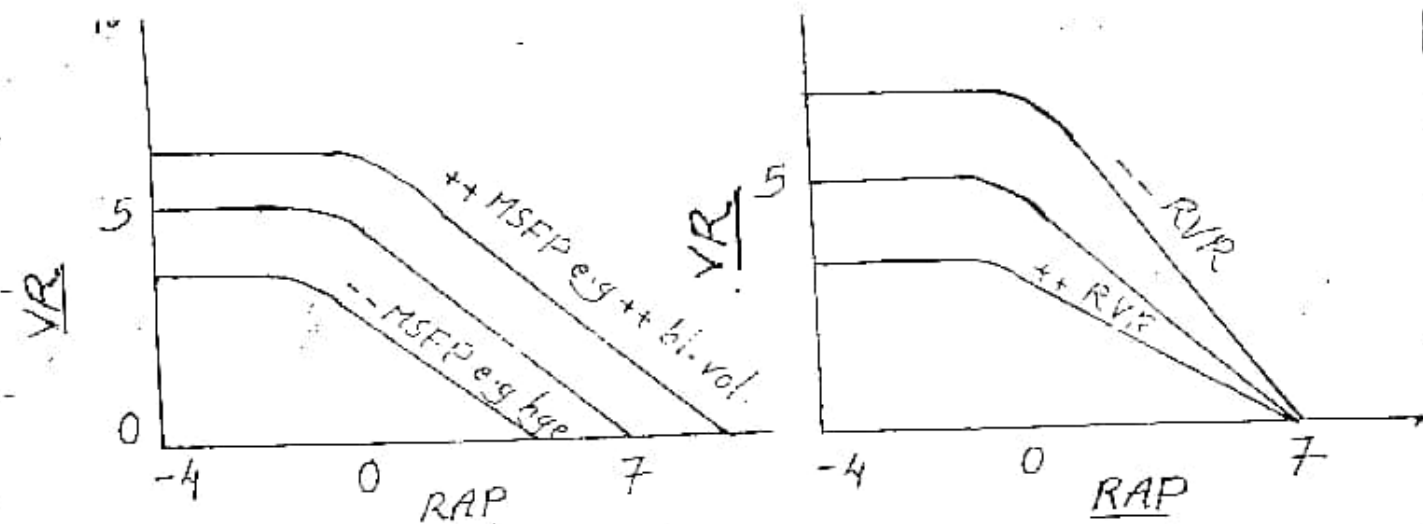
Unipolar limb leads



Augmented unipolar limb leads
same configuration
potential is 50% more.



Cir.



3) RVR

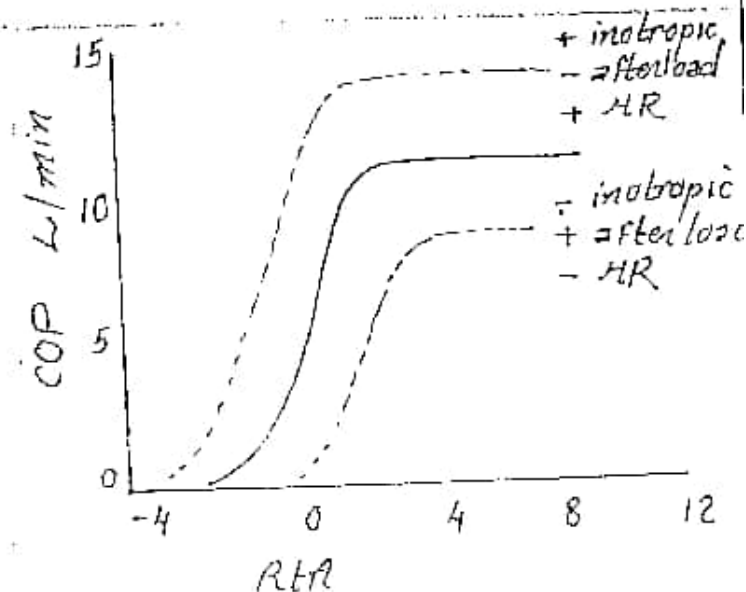
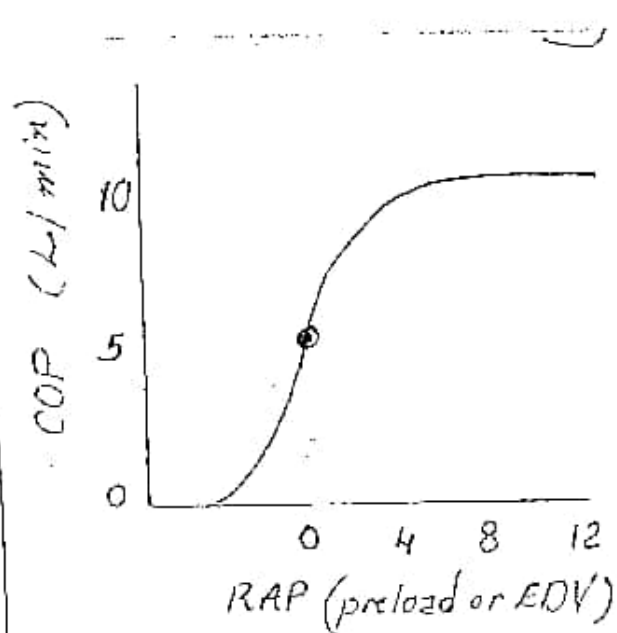
$$F = \frac{\Delta P}{R}$$

$$R = \frac{R}{\Delta P} = \frac{MSFP - RAP}{VR} = \frac{7 - 0}{5} = 1.4 \text{ mmHg/L/min}$$

Cardiac output curve

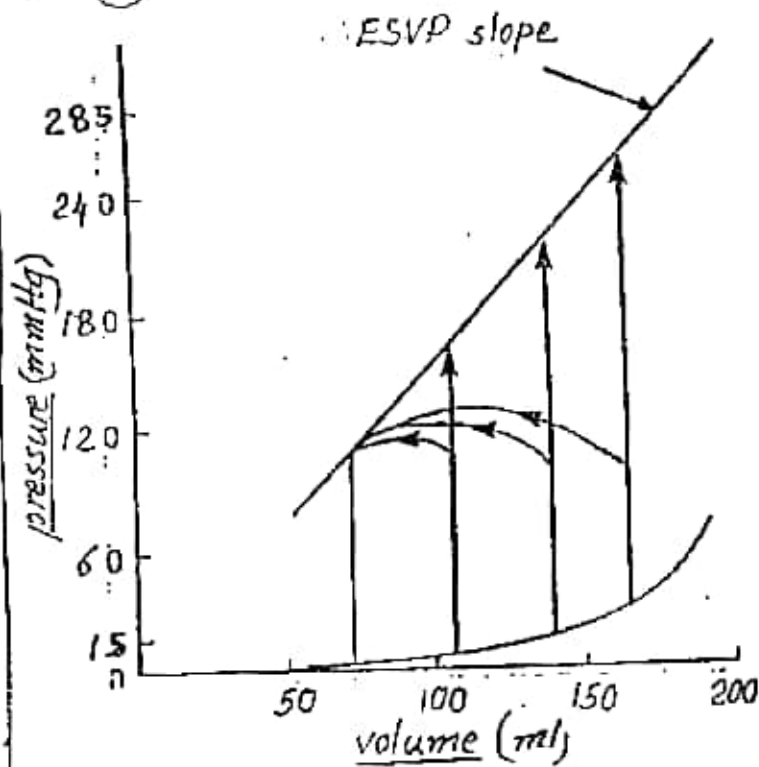
$COR \propto RAP$ within limits

Explanation: ++ RAP $\rightarrow$ ++ C. Filling $\rightarrow$ ++ EDV (preload)
 $\rightarrow$ ++ C contractility Starling law $\rightarrow$ ++ CO

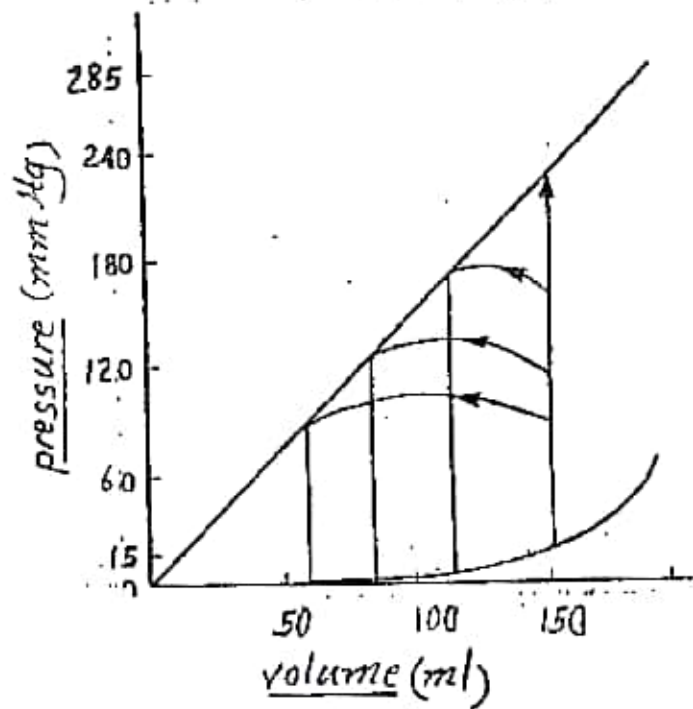


- 1 ++ EDV (preload) on SV. ESVP End Systolic Vent. Pressure
 - 2 ++ afterload on SV
 - 3 +ve and -ve inotropic stimuli on SV
- Effect of HR on COP

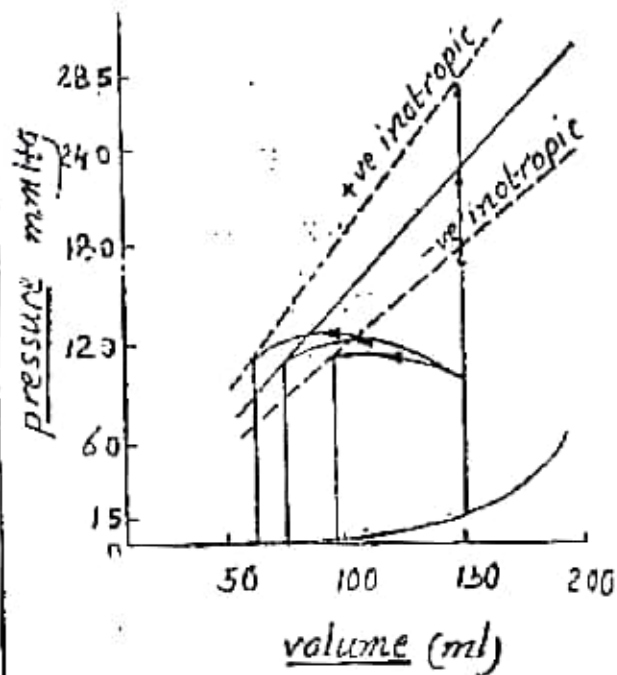
①



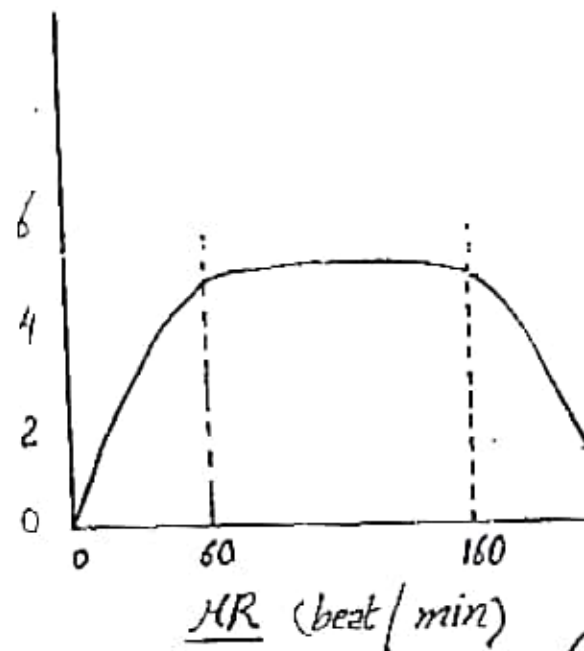
②



③



COP (L/min)



$$\text{Systemic vascular resistance} = \frac{105 (\text{diastolic P} + \frac{1}{3} \text{PP}) - 5 (\text{MSFP} - \text{RAP})}{20} = 5 \text{ L/min}$$

COP curve is influenced by 5 Factors :

- | | | |
|---|------------------------|--|
| 1 | <u>Preload</u> | Venous filling pressure |
| 2 | <u>Afterload</u> | Aortic p i.e ABP |
| 3 | <u>Inotropic state</u> | Contractility not dependent on [preload afterload] |
| 4 | <u>Heart rate</u> | |
| 5 | <u>Heart size</u> | Hypertrophy |

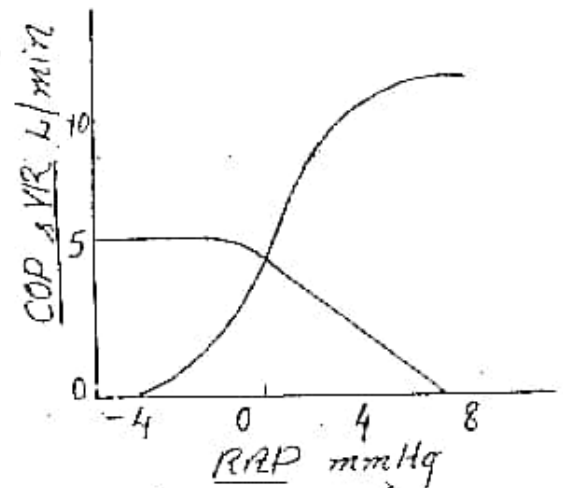
Equilibrium between VR and COP

At rest, 2 curves intersect at ..

at Rt atrial p 0-2 mmHg

Mechanism Starling law allows

COP to equal VR



Assessment of contractility

$$EF = \frac{SV}{EDV} = 0.6$$

Selected integrated responses

Supine to erect posture

Cardiac response to exercise

A HR .

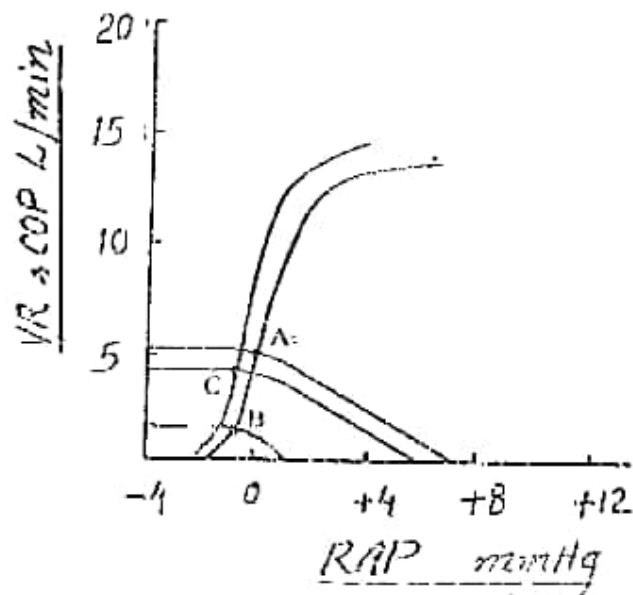
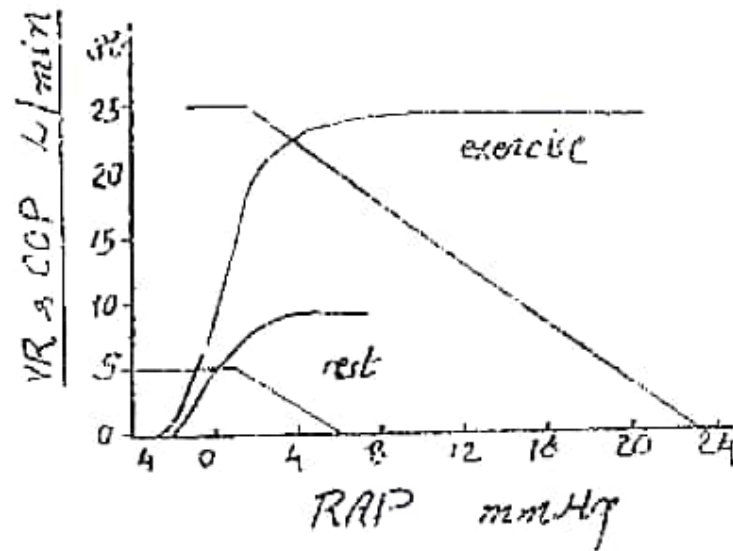
B Ventricular function .

C VR and COP .

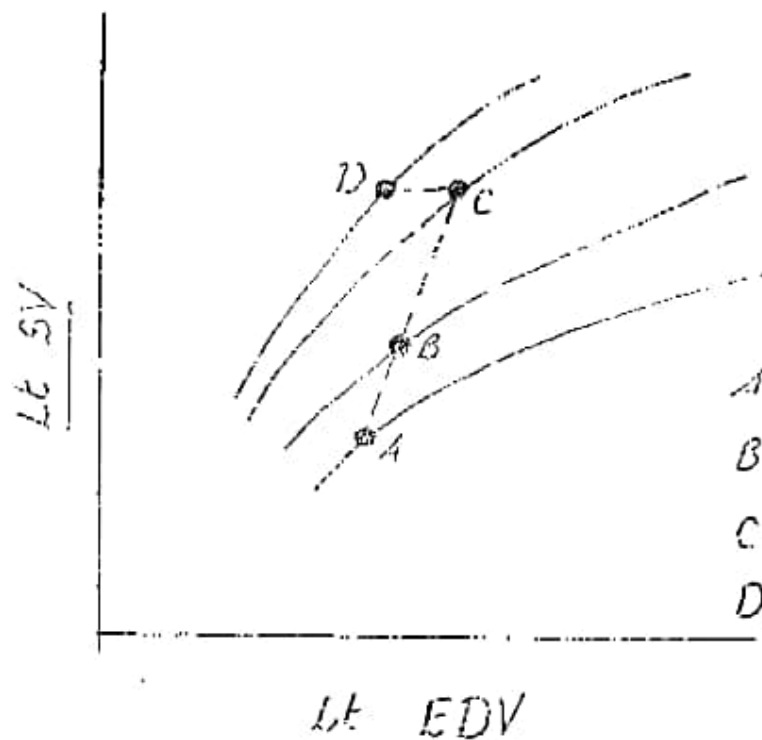
1 In untrained normal subjects .

2 In trained athletes .

Effect of exercise on equilibrium between CO and O₂

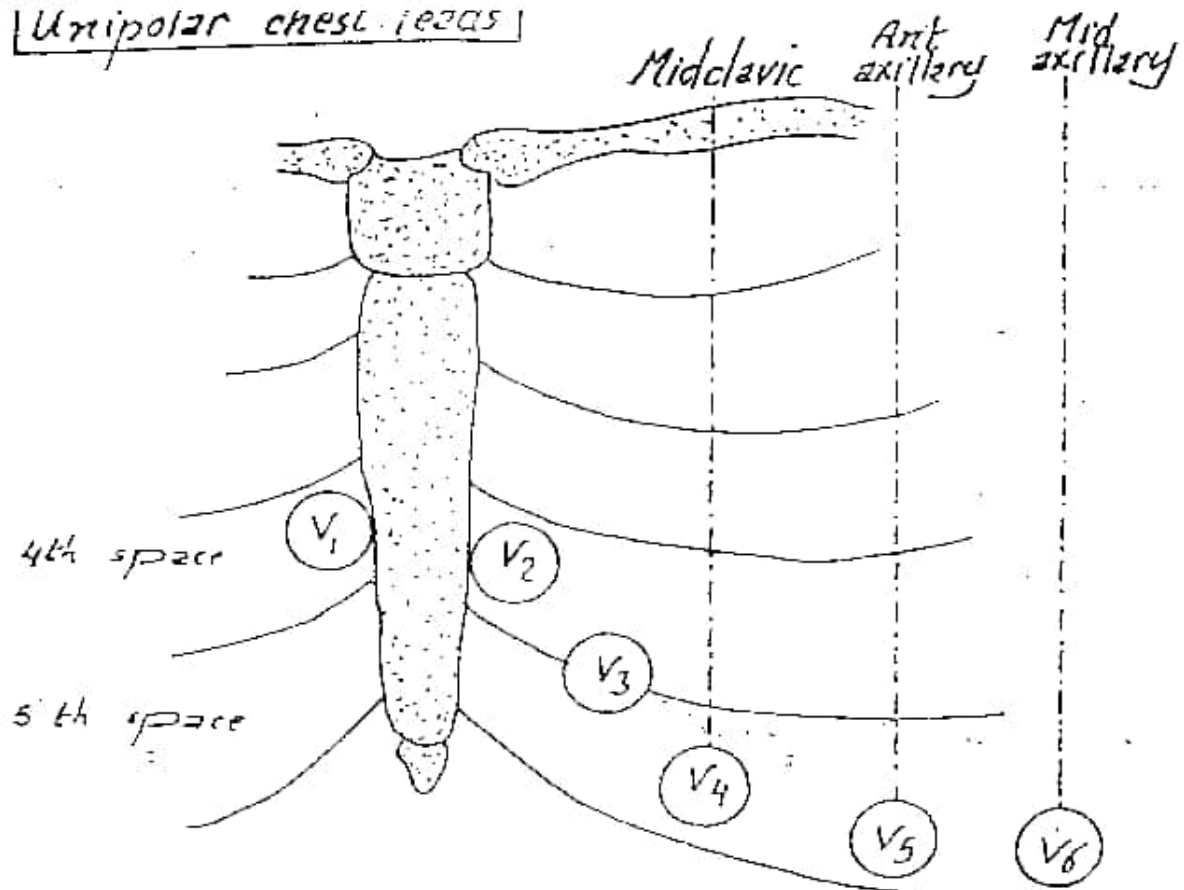


A control
B Upright tilting
C Compensation.

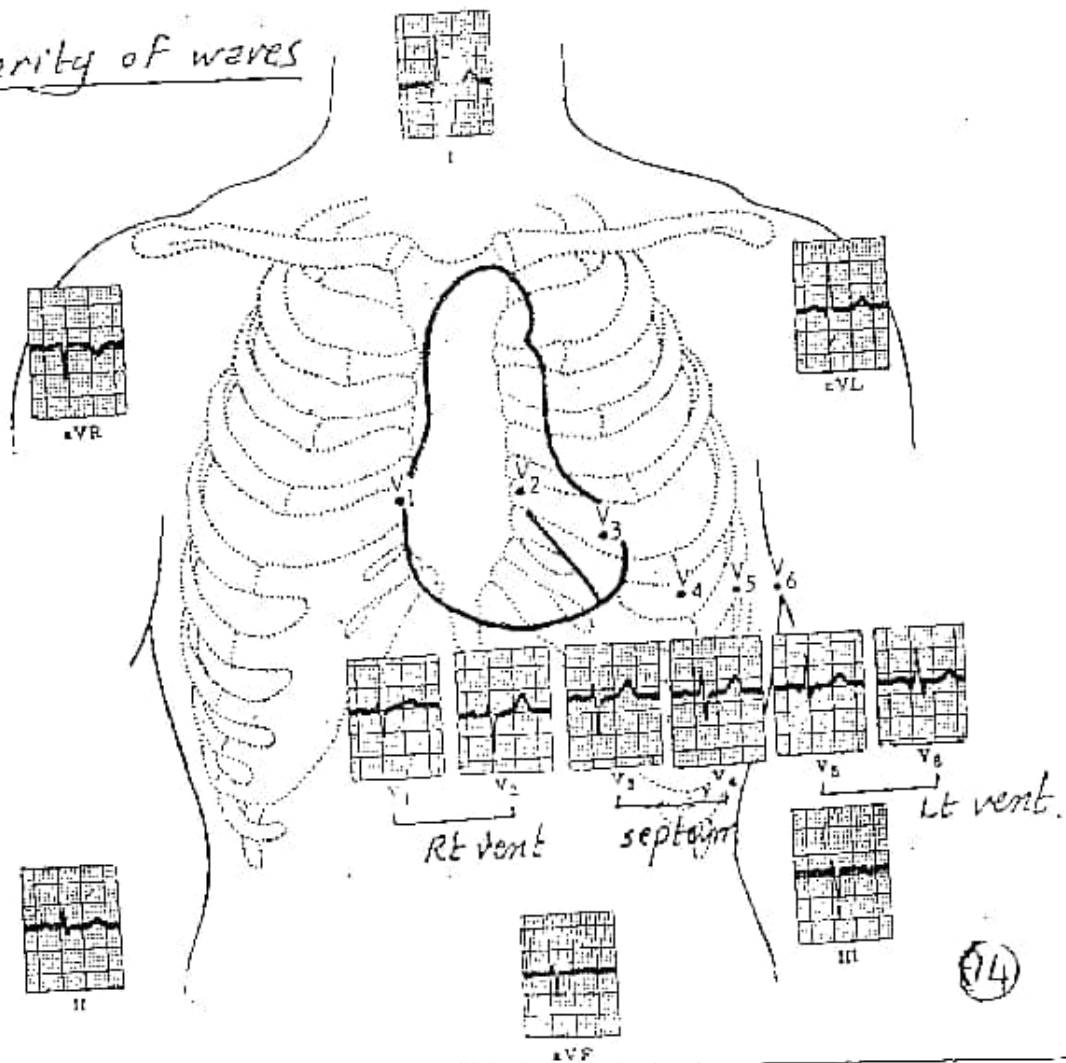


	HR	SV	COP L/min
A	70	80	5.6
B	120	100	12
C	150	120	17
D	170	115	19.5

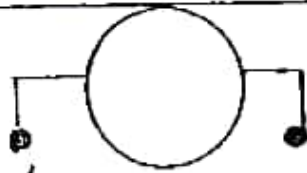
Unipolar chest leads



Polarity of waves



Leads



Bipolar limb leads

	<u>Exp</u>	<u>Exp</u>	
I	R	L	+ve electrode
II	R	F	+ve "
III	L	F	+ve "

Augmented unipolar limb leads:

	<u>Indiff</u>	<u>Exp</u>	
aVL	RF	L	+ve
aVR	LF	R	
aVF	RL	F	

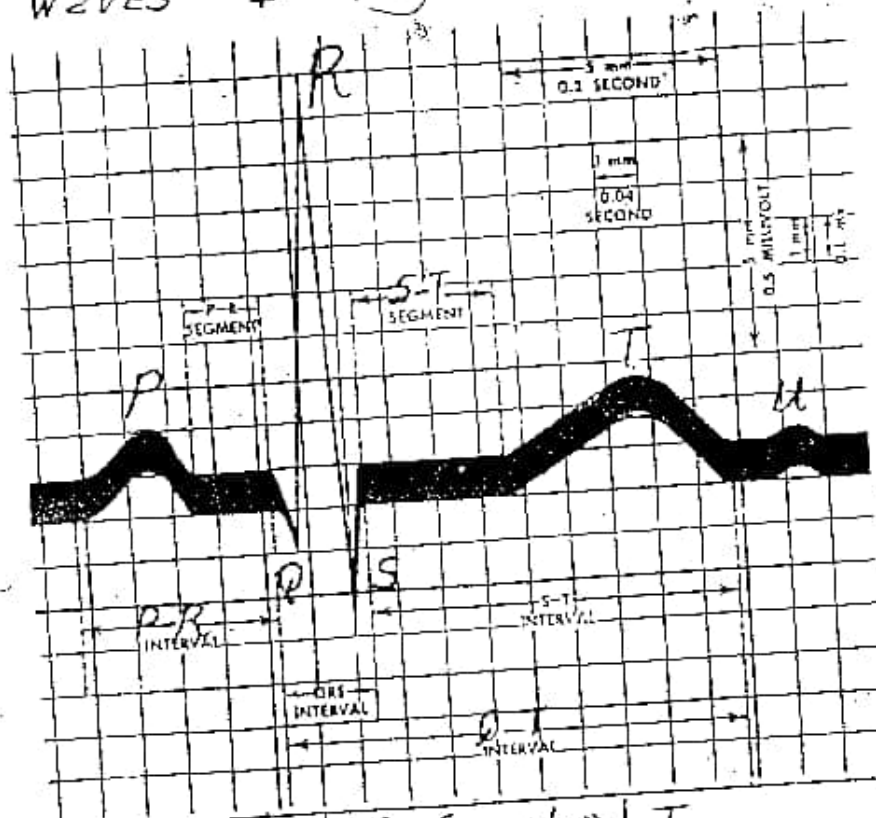
Unipolar chest leads

	<u>Indiff</u>	<u>Exp</u>	
V ₁	RLF	RL	4th space parasternal
V ₂	RLF	t	4th space parasternal
V ₃	RLF	t	Between V ₂ & V ₄
V ₄	RLF	t	5th space mid clavicular
V ₅	RLF	t	5th space ant. axillary
V ₆	RLF	t	5th space mid axillary

Cir

Normal ECG

Waves + Segments = Intervals



WAVES	P	QRS (complex)	T	U
<u>Represents</u>	A. depol.	V. depol	V. repol.	Papillary m repol
<u>Duration</u> (width)	0.08 - 0.15 i.e 80-100 msec	0.08 second	0.16 sec.	0.05 sec.
<u>Voltage</u> (height)				
Limb leads	0.25 mV	1 mV	> 0.5 mV	usually not recorded.
Chest leads		3-4 mV	> 1 mV	
<u>Shape</u>	1st half for RA atrium end half for Lt atrium	V1 V2 small R Large S V5 V6 large R small S	slightly rounded asymmetrical	
<u>Direction</u>	Inverted in aVR	R always +ve Q -ve before R S -ve after R	Inverted in aVR	

- A repol : masked by QRS complex & very low voltage
- SAN, AVN, AVB & Purkinje fibres : not recorded (small mass of tissue)
- Q wave < $\frac{1}{4} R$ if more than $\frac{1}{4} R$ = myocardial infarction
- appears in Lead I, aVL & aVF, V5 & V6
- T wave (repol) in the same direction of R wave (depol)
- Last part to be depol (epicardium) is the 1st to be repol.

Segments

• PR segment

- Measurement

- Represents

- Normal

end of P to
beginning of QRS
conduction in AV node
0.08 to 0.1 second



• ST segment

- normal
isoelectric

same level of
T-P line

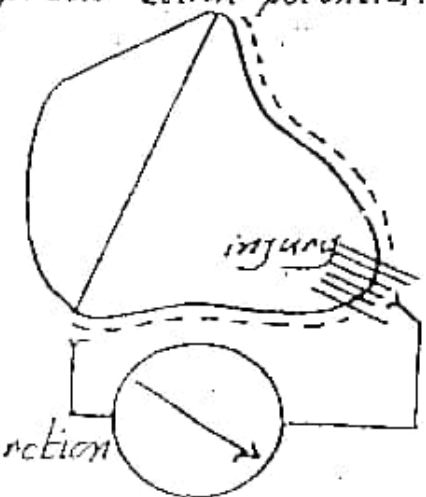
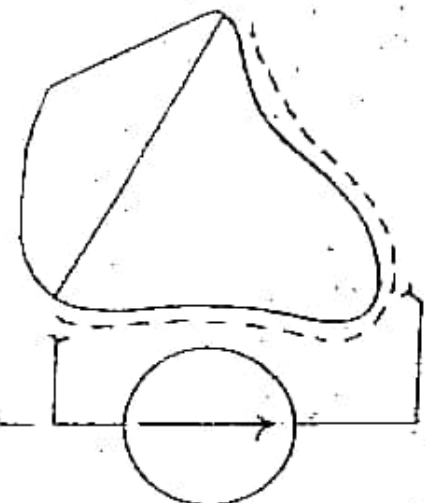
0.32 sec

- coincides with

- Injury

elevated by
2-3 mV

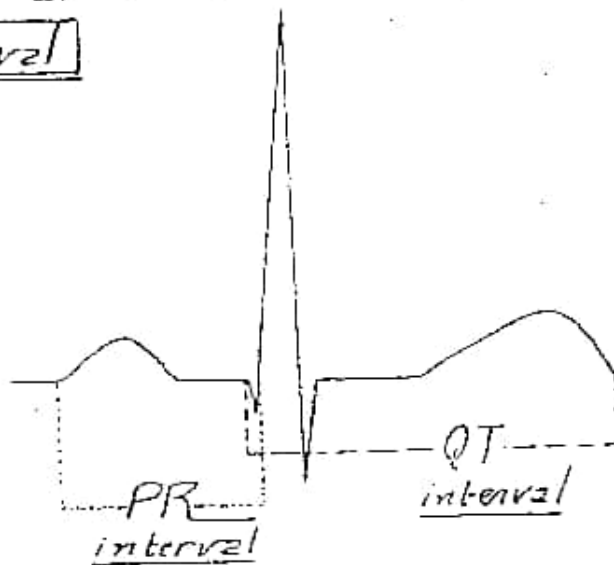
plateau of monophasic action potential.
i.e. 0.32 sec.
i.e. vent repol.



= Recent myocardial infarction

Intervals waves + segments

● P-R interval



Measurement

Beginning of P
Beginning of R

Represents

A. depolarization
Conduction in AVN & AVE

Normal

0.16 - 0.20 sec (average 0.18 second)

Prolonged

A-V Block
Atrial enlargement
Vagus stimulation

Shortened

A-V nodal rhythm
Symp. stimulation

Q-T interval

Measurement

beginning of QRS complex
end of T

Represents

Ventricular depol + vent repolariz

Normal duration

0.40 second

Prolonged

Hypocalcaemia